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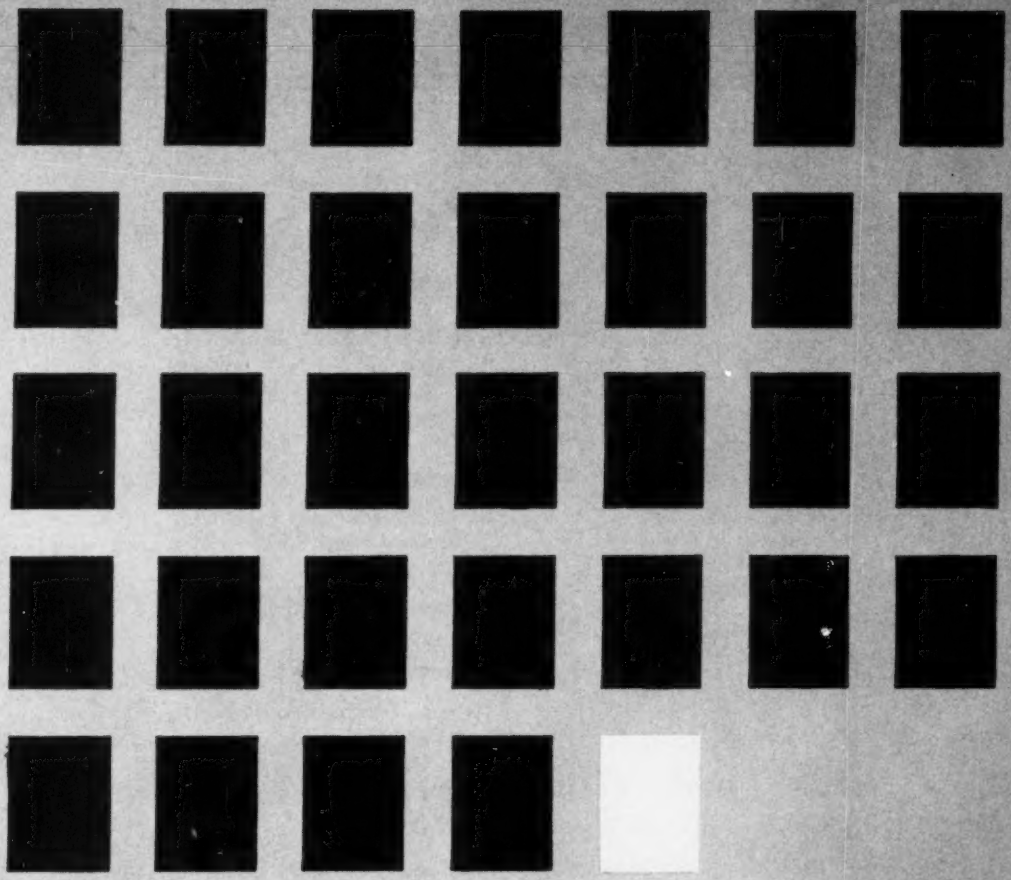

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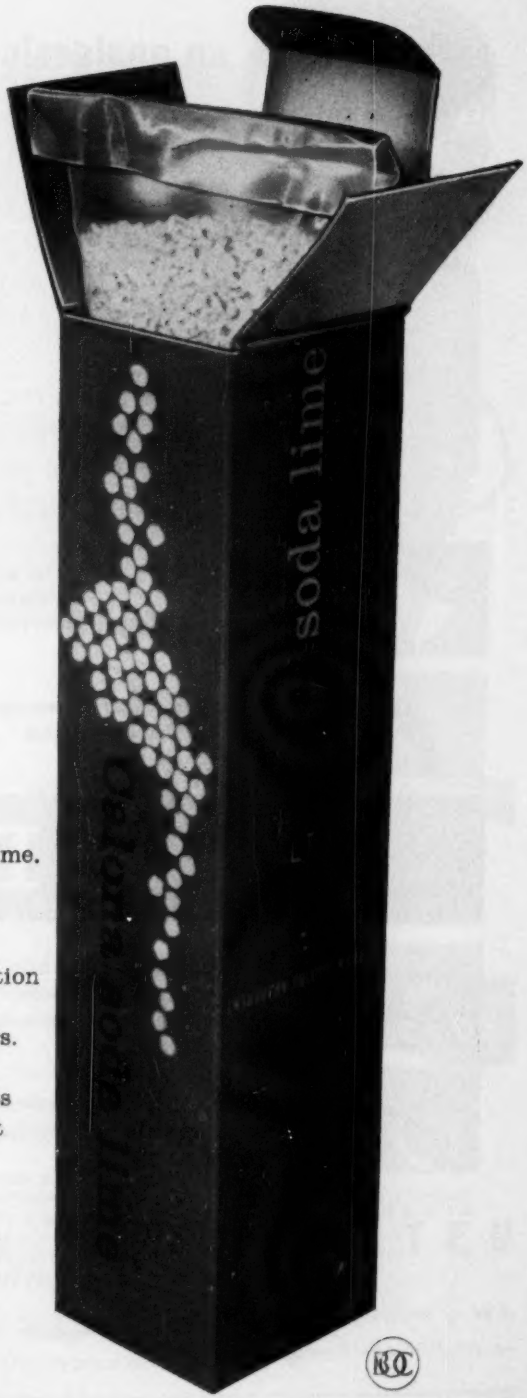

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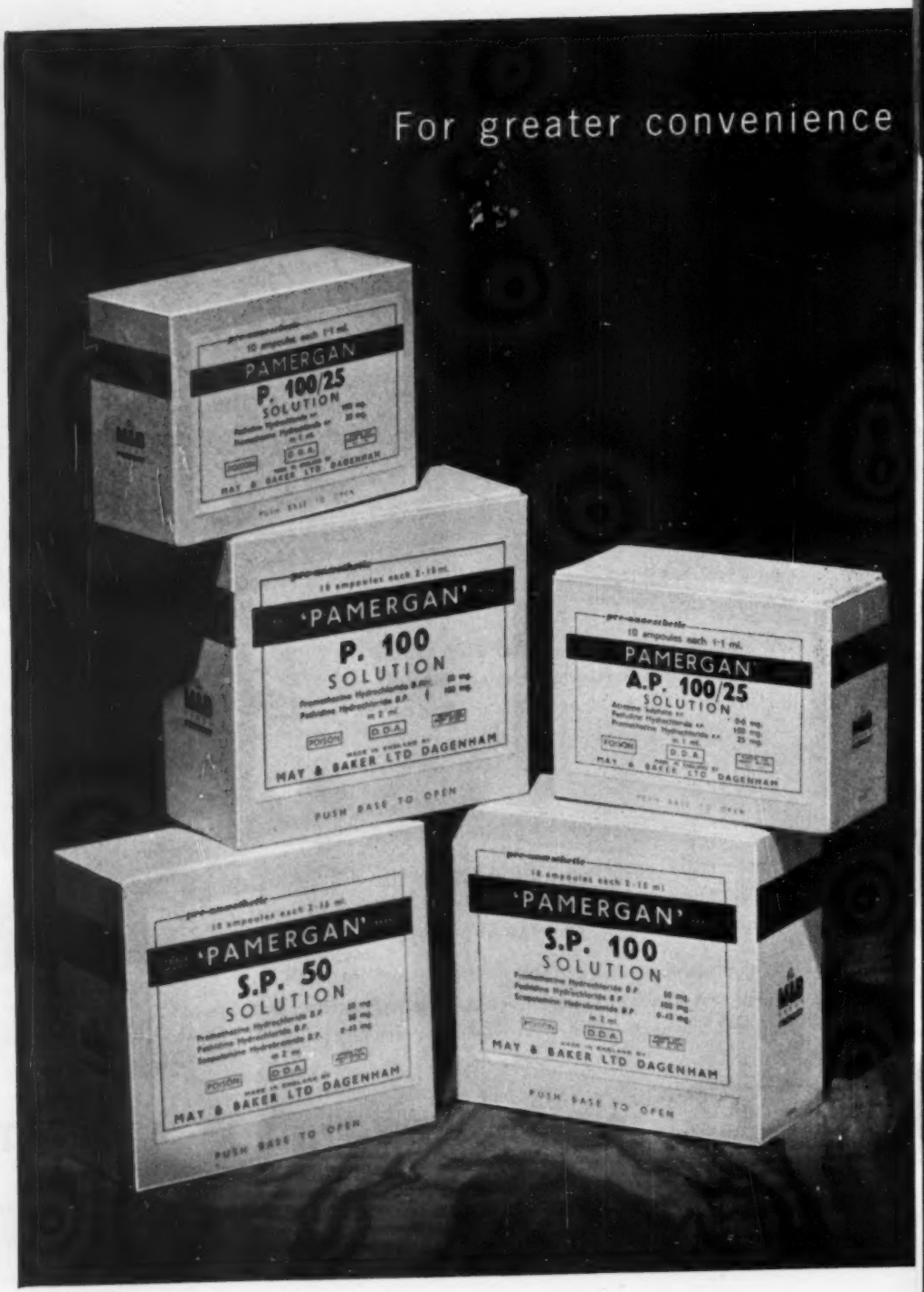
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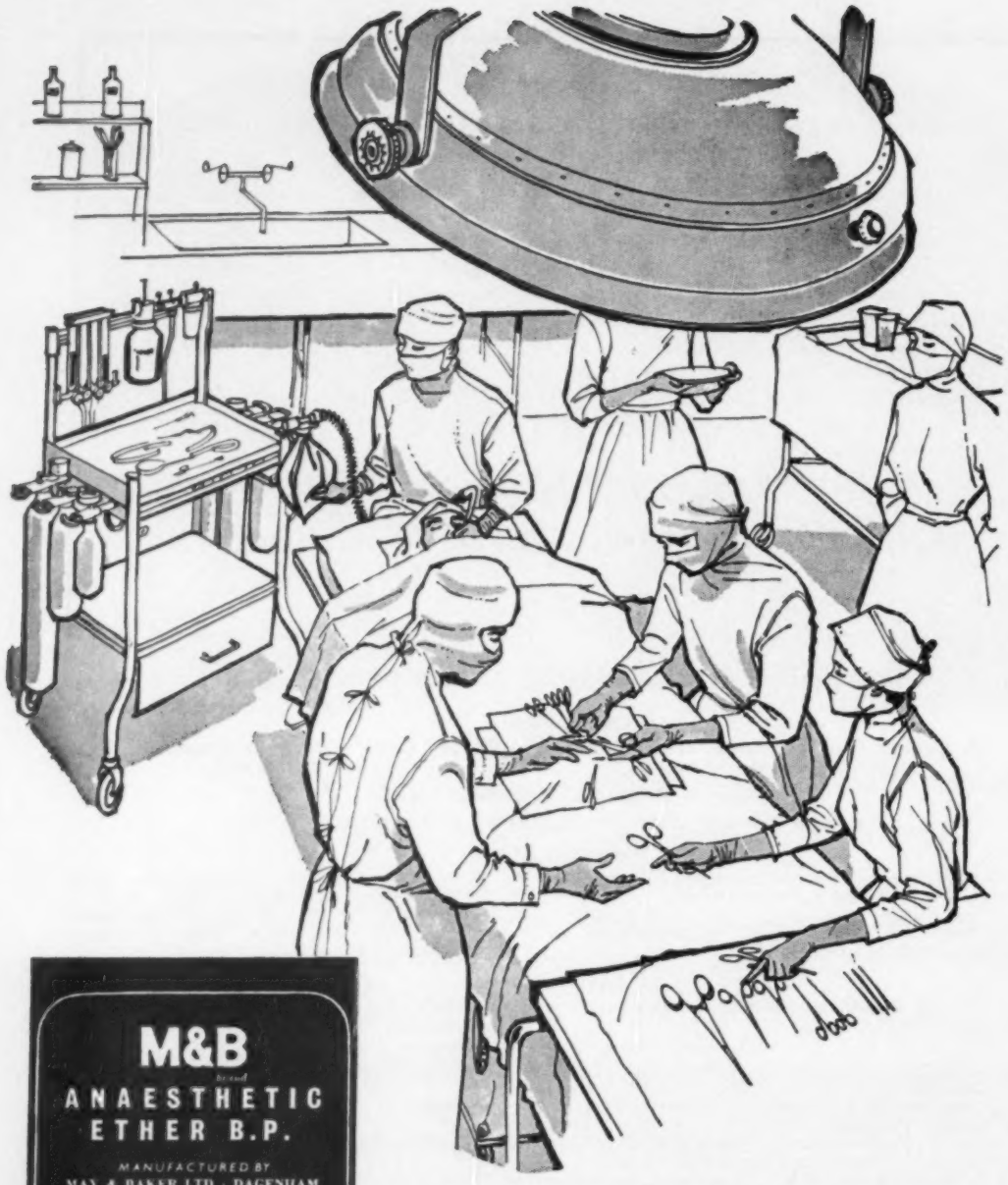

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BRITISH JOURNAL OF ANAESTHESIA

NOVEMBER 1960 VOL. XXXII, No. 11

EDITORIAL

PHYSICIANS AND THE ANAESTHETIST

FIFTY years ago the "chloroformist" was a physician who used one drug and one technique for all manner of operations. An almost certainly apocryphal story has it that a surgeon once arrived to operate upon a patient in a private house and found the physician and patient in what appeared to be mortal combat: during the second stage of anaesthesia the physician had become pinned down upon the floor with the patient uppermost! As a consequence of this and similar occurrences and of the general inferior status of the "chloroformist", physicians for the most part were not attracted to this rough-and-tumble job and sought a gentler art. Others, more intrepid, forged ahead to develop a specialty with a glorious but difficult future.

Progress was sometimes halted by authority, for which there is no place in science; this occurred, for example, when an eminent obstetrician urged that chloroform was safer than ether. It is not many years ago that "authority", generally in the shape of a physician, did not hesitate to state that this or that patient was "fit for gas and oxygen only". On the other hand, there has always been a danger that the practising anaesthetist, in the development of the technical skill upon which his living depends, would become more and more a mere technician. A gulf has thus tended to widen between the anaesthetist and the physician.

Because of the contribution that anaesthetists have made towards the understanding of respiratory physiology and the handling of the patient with respiratory disorders, this gulf has narrowed in recent years, and there is no doubt that at present both physician and anaesthetist can benefit from each other's knowledge and skill.

There are several ways in which physicians can help anaesthetists. The diagnosis may require confirmation or the severity of the disorder may have to be assessed. In the light of these decisions, further medical treatment may be required before operation is carried out. A better understanding of the results of disease upon the functions of systems and organs should also help the anaesthetist to plan his procedures more rationally.

Brit. J. Anaesth. (1960), **32**, 514

NEUROLOGICAL ASPECTS OF ANAESTHESIA

BY

D. TAVERNER

Department of Medicine, University of Leeds, England

THE central nervous system is more easily damaged or destroyed by deficiencies of anaesthetic technique than any other tissue in the body. Yet, paradoxically, it usually matters little to the anaesthetist whether his patient's nervous system is healthy or not. The margin of error is slight, even in health, because neurones are so readily injured by anoxaemia. First class anaesthesia is a better safeguard for the patient than neurological expertise.

Even so it is desirable that the anaesthetist should be aware of certain special dangers which may ensue in patients of several kinds. The presence of raised intracranial pressure, injuries to the spine, cerebro-vascular disease, bulbar palsy, myasthenia gravis and some rare metabolic diseases, involves unusual risks, which can to some extent be foreseen and avoided. It is clear that the anaesthetist can hardly be expected to diagnose most of these conditions on the anaesthetic trolley; some of them may tax the resources of an experienced neurologist. He will normally expect to be warned of their presence, but if he can visit the patient before operation he may well recognize the mental retardation of some patients with cerebro-vascular disease or the drooping lids and mask-like face of myasthenia. Thirty seconds with an ophthalmoscope should serve to exclude gross papilloedema resulting from high intracranial pressure.

If we assume that the neurological diagnosis is established it is evident that the anaesthetist still needs to know the extent and severity of the disease if he is to act appropriately. His approach will be governed by an understanding of the potential dangers and implications of the condition present. The purpose of this article is to present information about present views and beliefs in this field. Extensive experience of anaesthesia in almost every conceivable neurological condition has now been accumulated by surgeons of all kinds, physicians, psychiatrists and, of course, anaesthetists. The great bulk of neurological disease is unaffected by properly conducted general anaesthesia and need not be seriously considered by the anaesthetist. Although precise evidence is lacking it would seem to be a reasonable precaution to avoid spinal anaesthesia in patients with neurological disease because rare and unpredictable sequelae can occur even in those with good health.

There remain for consideration the neurosurgical patients, those with cerebral ischaemia, myasthenia and sufferers from certain rare metabolic disorders.

Neurosurgical problems.

Trauma to the skull and spine may present the anaesthetist with dangerous and hidden problems. Injury to the spinal column with fracture or dislocation of the vertebrae may leave the blood supply of the spinal cord in such a precarious state that any careless manipulations, especially hyperextension of the neck, may lead to necrosis of the cord, with irreparable paralysis and sensory loss. Sometimes less severe damage results which may cause severe distress or disability because of subsequent weakness, sensory loss or inadequate bladder control. The anaesthetist should be always aware that similar damage may follow injudicious manipulations in patients with spinal tumours or with the cervical spondylosis which is so common in the middle-aged and elderly.

Patients with severe head injuries usually suffer brain stem damage with coma, stertorous breathing, disorganization of pharyngeal movements and loss of the swallowing reflex. Blood, vomitus and secretions accumulate in the pharynx and are readily inhaled. The struggling, restless, noisy, cyanotic patient deteriorates steadily. The high

venous pressure raises the intracranial pressure, so increasing the degree of brain damage and creating a vicious circle. The essential manoeuvre here is emergency tracheal intubation with a cuffed tube.

Anaesthesia for routine neurosurgery is not unusually hazardous unless the intracranial pressure is high. Correct preparation of the patient by suitably propping the head and shoulders to avoid neck vein constriction, sedation and regular, easy defaecation are simple, effective precautions which may be overlooked. In some patients, preliminary ventriculostomy, under local anaesthesia, with gradual drainage of c.s.f. for two or three days, simplifies the operation. Tracheotomy with tracheobronchial suction, and spraying the carina with local anaesthetic to prevent coughing, are valuable preparations before certain urgent major operations.

The main danger in the presence of raised intracranial pressure is a sudden, severe, further rise of pressure from coughing, anoxaemia, respiratory straining and struggling. This can be avoided by expert anaesthetic technique but the intravenous injection of 50 per cent sucrose gives a helpful, transient reduction of intracranial pressure for 10–30 minutes. After the dura has been opened a sudden rise in the intracranial pressure may cause uncontrollable bulging of the soft brain with bruising, laceration, herniation and even extensive infarction. The danger can be reduced by the intravenous injection of urea at the start of the operation. This produces marked shrinkage of the brain for three or more hours, which covers the potentially dangerous part of the operation. Some anaesthetists prefer the added security of total neuromuscular block during this period.

Emphasis on the intracranial pressure must not distract attention from the danger of an unexpected drop in blood pressure during any surgical procedure. Cerebral ischaemia and infarction may then occur with startling speed, especially in older people with damaged, narrowed, arteries in the head and neck. Anaesthesia in the sitting position, whether for middle or posterior fossa exposures, or for simple dental extraction, is especially dangerous. The blood pressure must be continuously monitored in such circumstances so that rapid counter measures, by posture and drugs, can be undertaken. The ever-present risk of air embolism in this position must not be overlooked.

Variations in anaesthetic technique may have important consequences for the patient because changes in respiratory rate and tension of oxygen and carbon dioxide profoundly modify the cerebral circulation. Excess of carbon dioxide or deficiency of oxygen produce marked vasodilatation with swelling of the brain, fall in cerebral vascular resistance and considerable increase in cerebral blood flow. Conversely, artificial hyperventilation or excess of oxygen (80–100 per cent) constricts the brain and its vessels and much reduces the cerebral blood flow.

Surgical anaesthesia itself reduces brain metabolism by 30–40 per cent and its oxygen consumption is theoretically less than in the waking state. To a certain extent the brain has self-protective mechanisms against hypoxia, but these may lead to damage from brain swelling when the intracranial pressure is high or the dura is open. Moreover, the normal mechanisms of vasodilatation are inoperative in patients with severe hypertension or cerebral arteriosclerosis and their brains are readily damaged by a degree of hypoxia which would be harmless in the young.

Primary muscular diseases.

Primary disorders of muscle are rare and do not usually concern the anaesthetist. Many of the sufferers develop respiratory impairment in the later stages but this can readily be controlled by the anaesthetist in the rare instances when surgery is needed. It is worth mentioning that the heart may be involved in acquired muscular disease, such as polymyositis, and in the muscular dystrophies. The progressive muscular dystrophies are a clinically complex group of conditions in which primary muscular degeneration with progressive weakness and wasting occurs in various patterns. They are genetic in origin and the diagnosis is relatively simple when the signs are well developed. The heart is involved in about half the patients with tachycardia, extrasystoles, various electrocardiographic abnormalities and myocardial fibrosis. Cardiac failure may develop and sudden death has been reported several times (Zatuchni et al., 1951). The special case of dystrophia myotonica is discussed later.

Neuromuscular transmission.

The selective permeability to ions of the limiting membrane of a muscle fibre causes it to be polarized so that the inside is 80–100 mV negative with respect to the outside. When a nerve impulse reaches the specialized junction between the nerve fibre and the muscle fibre—the motor end plate—a jet of acetylcholine is released. The acetylcholine reacts with protein molecules (cholinergic receptors) in the postjunctional membrane. The configuration of the receptors changes and this alters the permeability of the membrane so that a rapid inward rush of Na^+ ions and an outward flow of K^+ ions occur. This sudden flow of ions depolarizes the membrane and, once started, a wave of depolarization sweeps along the surface of the muscle fibre. An electrode outside the fibre records these electrical changes as a wave of negativity—the action potential—which is propagated over the whole muscle fibre. In some way not yet understood this electrical wave triggers off the contraction of the muscle protein and produces a muscle twitch.

Depolarization lasts only 2–3 m.sec because the acetylcholine released is rapidly hydrolyzed to choline and acetic acid by the enzyme cholinesterase which is always present in the motor end plate. The muscle fibre membrane is then rapidly repolarized and a further twitch can result from another nerve impulse which arrives about 10 m.sec after the first. Such rapid rates of firing would produce a sustained muscular contraction or tetanus.

Neuromuscular block.

Neuromuscular transmission can be modified in various ways. Alterations in the amount of Ca^{++} ions and botulinus toxin both affect the release of the transmitter (acetylcholine), while changes in the concentration of Na^+ and K^+ ions inside and outside the muscle fibre can profoundly affect the responsiveness of the end plate. Substances like d-tubocurarine and gallamine, which compete with acetylcholine for the cholinergic receptors on the junctional membrane, passively block transmission without causing depolarization (competitive or nondepolarization block). Other drugs such as decamethonium and succinylcholine cause prolonged depolarization of the junctional membrane so that further response to nerve impulses is impossible (depolarization block). There is a marked difference between species and in many instances mixed types of block may develop.

There are differences between the two kinds of block which may be clinically significant, especially when the block is partial. Nondepolarization block is increased by tetanic stimulation at rates of 50–100/sec but depolarization block is unaffected. Conversely, depolarization block is increased by the parenteral injection of anticholinesterase drugs, such as neostigmine or edrophonium, which counteract nondepolarization block. Depolarizing agents such as decamethonium are normally additive in their action, yet they oppose the activity of nondepolarization blocking agents such as d-tubocurarine.

Myasthenia gravis.

In spite of its rarity myasthenia gravis is by far the most important muscular disease for the anaesthetist because of its relationship to the neuromuscular blocking agents so commonly used in modern anaesthesia. It is a chronic, often progressive, disease of unknown aetiology in which variable weakness of voluntary muscle occurs after use. Most commonly the muscles responsible for eye movements, expression, chewing, swallowing or talking are involved in varying degree but the trunk muscles may also be affected. This sometimes causes respiratory failure. Although the effect of fatigue is obvious and the patients deteriorate during the day, it is striking that weakness will appear after a short mild effort which would not disturb the normal person, and a brief rest may cause temporary improvement. Later, however, permanent weakness or even total paralysis unaffected by rest, may supervene. In some patients, sudden and unexpected respiratory failure may occur after unusual exertions.

The disease usually affects adults between 20 and 50 years of age and there are two main forms. One is steadily progressive and is usually associated with a tumour of the thymus gland; the prognosis is poor even if the gland is removed surgically (Keynes, 1954). The commoner form is a relapsing, remitting, unpredictable disorder which may persist for many years and is fairly well controlled by drug therapy or thymectomy or both.

Neuromuscular transmission in myasthenia gravis.

The exact nature of the transmission defect in myasthenia gravis is not yet clear but it has many of the features of nondepolarization block. It is improved by anticholinesterase drugs and made worse by tetanic stimulation and by nondepolarizing blocking agents like d-tubocurarine. It is now widely believed that the essential defect in myasthenia is an abnormal response of the motor end plate to acetylcholine, but its cause and nature are unknown. At times myasthenic patients may suddenly develop a "cholinergic crisis", when they no longer respond to anticholinesterase drugs because depolarization block has developed. This is a dangerous state requiring specialized care.

Clinical implications.

The diagnosis of myasthenia gravis is often overlooked for long periods and clinical awareness of the possibility is the chief safeguard. Once the condition has been suspected the simplest confirmation is by the injection of anticholinesterase drugs. The most efficient method is the use of edrophonium (Tensilon) because it reverses the myasthenic block in about 30 seconds. This is one of the most dramatic experiences in medicine, although depressing for the unwarned patient because the effect disappears again in 10–15 minutes. The usual technique is to inject 2 mg of edrophonium intravenously and, if there is no response within 30 seconds, a further 8 mg is then given. In the untreated myasthenic patient some return of power is noted with the small dose and full, temporary, return of power follows the larger dose. In normal patients, those without myasthenia and in those whose myasthenia has been adequately treated, muscular fasciculation, intestinal colic, salivation, vomiting and diarrhoea may occur but any muscular weakness is unaffected. Very occasionally, false positive results have been noted in polymyositis and certain muscular dystrophies. Myasthenic patients who have developed cholinergic crisis have depolarization block and are therefore made worse by anticholinesterase drugs such as edrophonium. The edrophonium test is most useful in assessing the adequacy of treatment in those with myasthenia and it can be repeated as often as necessary because of the short active period of the drug.

Anaesthetic management.

The essential points in the anaesthetic management of myasthenia gravis are the maintenance of adequate alveolar ventilation, by artificial respiration, if necessary, and the avoidance of postoperative paralysis due to depolarization block (cholinergic crisis). Before operation the serum electrolytes should be estimated and any deficiency of K^+ corrected, because hypokalaemia increases neuromuscular block; a level of 4–5 m.equiv/l. is optimal. The adequacy of control by anticholinesterase drugs should be checked by the use of the edrophonium test and the proper dose of anticholinesterase should be given parenterally before operation. If depolarization block (cholinergic crisis) is already present surgery should usually be postponed until the patient has been given an opportunity to improve after stopping anticholinesterase drugs. Respiratory function should also be tested by measuring the vital capacity, the forced expiratory volume (1 second) and the maximum breathing capacity. Marked deficiency in respiratory function is an indication of the need for special care (Bergh, 1953).

Minimal pre-operative medication is best followed by endotracheal cyclopropane and oxygen (Mathews and Derrick, 1957); the thiobarbiturate drugs are said to be liable to produce prolonged respiratory depression. Relaxants are not usually needed but, if one must be used, suxamethonium is to be preferred because it seems to act upon myasthenic end plates in the same way as in normal subjects and it is short acting. Nondepolarizing blocking agents such as d-tubocurarine and gallamine should never be used because myasthenic patients are hypersensitive to them, although, surprisingly, they are often resistant to decamethonium, a depolarizing agent (Churchill-Davidson and Richardson, 1955). Postoperatively, special care is required to maintain adequate ventilation and to prevent pulmonary complications by suction or bronchoscopy if secretions accumulate. An increase in anticholinesterase dosage may be needed for a time after operation and the efficiency of treatment should be checked repeatedly by the edrophonium test. Prolonged apnoea after operation should not be taken as an automatic indication for more neostigmine; if depolarization block has developed such treatment exacerbates it.

Dystrophia myotonica.

This hereditary disease is not very rare in its minor forms. The severe disease can often be recognized at a glance by the combination of cataract, expressionless face, severely wasted sternomastoid muscles and frontal baldness. There is diffuse limb weakness with a characteristic failure to relax after muscular contraction. The anaesthetist who shakes hands with every patient can hardly fail to remark on the dystrophic patient's failure to let go. Percussion myotonia—a sustained localized contraction of a tapped muscle—is commonly found. Bradycardia, prolonged PR interval and QRS abnormalities have been described.

The disease is a primary abnormality of the muscle fibre and until recently it was believed that such patients were abnormally sensitive to thiopentone (Dundee, 1952). A recent careful study (Kaufman, 1960) contests this and shows that the anaesthetic problems are similar to those of any patient with severe muscle weakness. Ventilation is apt to be impaired by the myotonia and postoperative respiratory obstruction is a particular danger because of the greatly weakened neck musculature. In such patients minimal premedication and expert anaesthetic technique with adequate ventilation are of greater importance than choice of agent.

Acute intermittent porphyria.

Choice of anaesthetic agent is, however, of great importance in this condition which is accentuated by any form of barbiturate. In fact, not a few patients are first diagnosed after an unnecessary operation using thiopentone or other barbiturate anaesthetic compounds.

Porphyria is an uncommon familial disorder affecting adults of either sex and is transmitted as a Mendelian dominant characteristic. The patients suffer paroxysms of abdominal pain with vomiting and constipation which are often regarded as abdominal emergencies. They may show neurotic of bizarre behaviour, and neuromuscular disorders consisting either of a polyneuritis or an ascending flaccid paralysis culminating in respiratory failure may occur. The urine turns dark red or black after standing for some hours because of the presence of porphobilinogen. Barbiturates must be entirely avoided because they exacerbate the condition and may cause circulatory collapse.

Potassium depletion.

Potassium depletion is not uncommon in surgical practice and may cause weakness or paralysis of muscle and hypersensitivity to neuromuscular blocking agents. There may be inadequate intake of K^+ or excessive loss by vomiting, diarrhoea or loss in the urine because of the excessive use of diuretic drugs such as chlorothiazide, potassium losing nephritis or primary aldosteronism. The rare condition of familial periodic paralysis is also often associated with low serum potassium values. Whenever potassium deficiency is suspected the serum level should be measured. Low levels can usually be corrected to the normal range of 4–5 m.equiv/l. by the use of oral potassium chloride.

CONCLUSION

Many neurological conditions from acute poliomyelitis to amyotrophic lateral sclerosis may be associated with bulbar palsy and respiratory weakness. They should present little difficulty to the experienced anaesthetist because he is well practised in the maintenance of adequate ventilation, oxygenation and circulation in the anaesthetized, paralyzed patient. Certain special problems have been discussed in this article but fundamentally, good anaesthesia is little modified by the prior neurological status.

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IMPORTANT FEATURES OF CHEST DISEASES

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THE modern anaesthetist has responsibilities which stretch far beyond the administration of anaesthetic drugs; he has always had to have more than a passing interest in the state of his patients' lungs, but in these days of thoracic surgery on an ever increasing scale, his interest has to keep pace with advances in the scope of this branch of surgery in particular. This is the main reason for writing what follows. It is manifestly impossible to do more than touch upon diseases of the chest at all completely in the course of a single article no matter how carefully written. The plan adopted here has had to be as general as possible, dwelling upon the important physical signs and symptoms rather than attempting descriptions of chest disease in any comprehensive way. Certain conditions have been selected for special mention, however, not only for their general application in considering pulmonary disease from a purely medical point of view, but also because they have special interest for the anaesthetist. It may be useful to realize that much of the training and practice of the anaesthetist can be used most effectively not only in the recognition of diseases of the lungs but also in their treatment. If what has been written here can bring this home to the reader and stimulate interest, he will find much that is satisfying in his work which perhaps he did not recognize before, and his patients and colleagues will benefit accordingly.

HISTORY TAKING AND SYMPTOMATIC ENQUIRY

A short history of the patient's illness in his own words is always required and it is usually desirable to ask a few leading questions to elucidate points in the history which the patient may have missed out or regarded as irrelevant. The important symptoms which may be used at first as a guide in this inquiry are:

Lassitude. Sleeplessness. Weight loss. Loss of appetite.

Cough (frequency during the day).

Sputum (volume, character and colour). Haemoptysis.

Breathlessness (constant, exertional or spasmodic).

Pain in chest or abdomen (duration, character and relation to movement and breathing).

Nasal symptoms. Attacks of sore throat. Changes in voice.

Alcohol and tobacco consumption.

Previous illnesses should be noted, especially recent ones and a family history obtained. In the case of infective diseases special inquiry should be made about contacts with others. Particulars of previous treatment are often important.

PHYSICAL EXAMINATION

Only the important points will be mentioned—it being assumed that the reader has not forgotten all he learnt as an undergraduate!

Inspection. General inspection of the patient is particularly directed towards any gross anatomical abnormality, such as kyphoscoliosis, evidence of respiratory paralysis, or such conditions as the barrel-shaped deformity of the chest. A good indication may also be obtained of any abnormality of breathing (frequency, regularity or depth); it should also be noticed whether both sides of the chest move equally and whether there is any abnormal swelling or contraction of the chest on either side. Evidence of respiratory obstruction may also be obtained by observing the behaviour of the intercostal spaces. Pronounced indrawing of these spaces during inspiration is a common sign of bronchiolar obstruction, especially in children. Recession of the supraclavicular fossae may also be noticed when the apices of the lungs are the seat of fibrosis; it is

also occasionally seen in those generalized forms of pulmonary disease which result in rigidity of the lung tissue.

In examining the skin, it should be borne in mind that many pulmonary diseases have cutaneous manifestations also, as for example tuberculosis, sarcoidosis and the reticuloses. More important to the anaesthetist, however, is the detection of cyanosis; he is better qualified than most of his colleagues to detect it, but a few points are probably worth emphasizing concerning the clinical appraisal of this important sign. The skin and mucous membranes owe their colour to the state of the haemoglobin in the blood of the subcutaneous capillaries; their temperature depends upon the speed of blood-flow through them. If for any reason the latter is slow and the capillaries are dilated, cyanosis will be apparent; this is due solely to reduction of the oxyhaemoglobin of the blood in the capillaries and is not a true reflection of the colour of the blood in the arterioles supplying them. This is the state of affairs when one's nose is "blue with cold". On the other hand in the case where the skin appears blue and is hot, central cyanosis is present; this can only arise when the arterial blood is under oxygenated. The best place to look for central cyanosis is not the face, lips or hands, which may be cold and exhibit "peripheral cyanosis", but to examine the buccal mucous membranes or tongue. Medd et al. (1959) state that the artificial light by which these are examined should not be too bright. With sufficient practice—checked by laboratory measurements of blood oxygen saturation—a very rough assessment of the degree of suboxygenation may be made. The deepest cyanosis is seen only in pulmonary disease; in heart disease—apart from congenital heart disease—the arterial oxygen saturation seldom falls below 85 per cent.

The hands and feet of the patient should be examined in order to reveal "clubbing" of the fingers or toes. Pain and swelling of the wrist and ankle joints (as well as the knees and elbows) is sometimes associated with clubbing of the fingers in pulmonary osteoarthropathy. An excellent description of this and its causes may be found in articles by Cudcowicz and Wraith (1957) and Coury (1960). The notion that it was usually associated with pulmonary disease which allows some arteriovenous shunting to occur in the lungs seems first to have been suggested by Mendlowitz (1938).

The upper respiratory tract should always be examined at this stage. Its condition has important bearings on the aetiology of segmental pneumonias which are not infrequent complications of the common cold, and may well have a similar significance in the production of post-operative chest infections.

Palpation. In addition to checking impressions already obtained during the first stage of the examination, palpation is valuable in fixing the position of the apex beat of the heart and in confirming the position of the trachea. The examination of the trachea is made a little easier if the patient's head is held in the position recommended for facilitating direct laryngoscopy (neck flexion and head extension). Any deviation from the mid-line may then be assessed by the relative ease with which the forefinger may be passed between the lateral surface of the trachea and the anterior border of the sternomastoid muscle.

Palpation of the abdomen forms an important part of the general examination of a patient with chest disease.

Percussion. Percussion of the chest was introduced as a method of physical examination by Auenbrugger (1761). This method if carried out systematically can yield most helpful data. It used to be customary to go to great lengths to define the surface markings of the dullness due to the heart and liver; this is not now necessary and was probably not so exactly defined in reality as might be supposed. As with all bedside methods of examination, practice will increase the precision of the observer, making fine differences in percussion note more easily detectable. The following details may be helpful.

It is useful to compare the sounds obtained by percussion over corresponding parts of the chest on either side in succession. Differences in sounds are thus more easily perceptible. It is important to be systematic; explore the front of the chest from above downwards and do likewise in the mid-axillary lines and over the posterior aspect of the chest. In examining the axillary area, get the patient to put his hand on the top of his head. The back is best examined with the patient

sitting forward with his hands grasped round his knees.

With regard to abnormalities of percussion note, there should be no difficulty in recognizing the stony dullness of a pleural effusion or the tympanitic note over a tension pneumothorax Hyperresonance, however, though often described, is in the opinion of the writer a more difficult sign to judge and still more difficult to interpret.

Auscultation. As in the case of percussion, skill in using the stethoscope comes only with practice and here again a systematic method is essential. Concentrated attention to the various elements which produce the sound is the basis of reliable interpretation. Sounds heard over healthy lung contain two distinct elements, the "vesicular" sounds which arise from the finest respiratory passages (and possibly the alveoli), and the bronchial sounds from the larger bronchi. The former are very faint and are often obscured by the latter. Sounds coming from the larger bronchi have one important characteristic; they do not occupy the whole of the respiratory cycle, the expiratory phase sounding shorter than the inspiratory. One of the changes heard in lobar pneumonia when the lung is consolidated is in this bronchial element of the breath sounds; the sound is said to be "bronchial" or "tubular", emphasizing the absence of the alveolar breath sounds. But the main characteristic of this abnormal sound is the fact that it occupies the whole of the expiratory phase and sounds just as loud as the inspiratory sound. Prolongation of the expiratory sound which is often heard in asthmatic patients is usually accompanied by an expiratory rhonchus or wheeze. For descriptive purposes, other sounds, the "added sounds", are included in accounts of auscultation. These are usefully grouped into the discontinuous sounds, "râles", and continuous or musical sounds, "rhonchi". These arise in the airways of the lung; râles are the result of interruption of the airstream by secretions, they are essentially bubbling noises whilst rhonchi are caused by turbulence of the airstream within the airways. These sounds vary both in pitch and intensity. Râles are variously described as "fine" or "coarse" and rhonchi are sometimes referred to as "wheezes". In conditions such as pulmonary congestion, the very fine râles heard best at the bases of the lung are called crepitations. Certain voice sounds can be transmitted to the chest wall and heard through a stethoscope; these are referred to as vocal resonance when the ordinary speaking voice is used by the patient, and whispering pectoriloquy when he whispers. Changes in these occur over lung tissue which is consolidated.

RADIOLOGICAL EXAMINATION

This aid to diagnosis is the most important one from the point of view of the chest physician. There are some who maintain that it has completely superseded the classical methods. This is only partly true, but it must be admitted that it makes exact anatomical diagnosis much more precise. Screening or fluoroscopy must be mentioned first and this can be of considerable help in the assessment of the functional state of the patient. In carrying out this examination it is important for the clinician to adapt himself to the darkness of the screening room. When this has been done a clear view of the chest can be obtained. Inspect the lung fields as a whole during quiet breathing. Note the movements of the thoracic cage and of the diaphragm, confirming any deviation of the trachea or displacement of the mediastinum. The lungs should light up uniformly during deeper breathing; any lack of uniformity is carefully noted. If pulmonary cysts are seen an attempt should be made to discover whether they are ventilated by noticing whether they enlarge during deep breathing. Paralysis of the diaphragm may not be obvious during quiet breathing unless it is complete, but it will be revealed at once if the patient sniffs sharply; in this case the affected dome will rise quickly instead of falling. This paradoxical movement is an important diagnostic sign. Opacities in the lung fields should be localized as accurately as possible by observing the position of the shadow in the lateral and oblique views of the thorax (obtained by rotating the patient as he stands behind the screen).

Standard radiographs of the chest should be available and give essential information. Their interpretation can scarcely be described here; appropriate textbooks should be consulted and every opportunity taken to practise this most

useful art by inspecting films under the guidance of a skilled radiologist.

SPUTUM EXAMINATION

Sputum should always be inspected and the daily output measured. Normal subjects do not produce sputum; but a patient who has even the mildest chronic bronchitis clears his throat or coughs frequently, expectorating an ounce or two of sputum in the course of a day. When the disease is more severe this amount may be as much as ten ounces and in such instances it is important to examine the appearance, which may be clear, colourless and viscid, consisting mainly of mucus mixed with a little saliva or it may be lumpy and very thick in consistency. The colour should be noted; yellow or green coloration of the sputum indicates active inflammatory disease with production of pus; the sputum may consist partly or entirely of pus. Blood may appear in the form of streaks of bright colour or the sputum may be diffusely stained; occasionally pure blood may be coughed up—a frank haemoptysis. Sputum may also contain altered blood which has a brownish colour—well described as rusty; this is commonly seen in acute pneumonia in the initial stages. Stained preparations of the sputum may be examined to confirm the presence of polymorphonuclear leucocytes or other cellular elements as well as pathogenic micro-organisms. Abnormal constituents, such as the Charcot-Leyden crystals, sometimes seen after a severe attack of asthma, may be noted along with the minute bronchial casts (Curschmann's spirals) characteristically seen in this illness.

Samples of sputum should also be sent to the laboratory for bacteriological examination.

RESPIRATORY FUNCTION TESTS

These tests are of considerable importance to anaesthetists and should in any case be a branch of clinical physiology with which he is especially familiar. A fairly full account of these has already been given in a recent textbook of anaesthesia (Prime, 1959). Suffice it to state here that the simple routine tests of lung function, namely the measurement of vital capacity and of maximum breathing capacity, are often of great help in giving objective information about incapacity, and that these are greatly increased in precision if supported by measurement of the arterial blood gases or the pulmonary diffusing capacity. This last named test demands special apparatus which is expensive, whilst the others can be done with the equipment found in most general hospitals.

VARIOUS LUNG DISORDERS

It is not possible within the available space to cover all the diseases of the lungs which the anaesthetist may come across in the course of his work. Instead, an account will be given of some aspects of collapse of the lung and pneumothorax, and of chronic bronchitis and emphysema. In the course of these discussions mention will be made of their bearing on the aetiology of many of the conditions likely to be met with by the anaesthetist. Fuller descriptions may be obtained by consulting any standard textbook, of which one of the best is by Coope (1948).

Collapse of the lung.

This term is applied to a diminution in the volume of the whole or part of a lung. It may be caused either by absorption of the gas contained in the lung, the result of complete obstruction of the airway (absorption collapse) or by compression of the lung by pressure from a space occupying lesion, such as a new growth in the adjacent parts of the lung (compression collapse). Collapse of a whole lung caused by obstruction of a main bronchus is usually referred to as massive collapse. In the case of absorption collapse the amount of lung tissue involved will clearly depend upon the magnitude of the airway obstructed and the alveolar territory it supplies. It usually is the consequence of aspiration of mucus, blood-clot or a foreign body and is often complicated by infection beyond the obstruction. It is a frequent cause of postoperative morbidity unless treated promptly. Pressure from outside the walls of the bronchi may also obstruct them; a common instance of this is seen in children where the cause is tuberculous infection and enlargement of hilar lymph nodes.

Symptoms and signs. These depend upon the extent of the lung involved and the rapidity of the collapse. Thus, massive collapse of the lung is usually a dramatic event with the development of pain in the chest, intense dyspnoea and "shock". Respiration is rapid and shallow and cyanosis is evident. The heart rate is rapid and a moderate

pyrexia may develop. Cough is usually stimulated. Physical examination will show that the mediastinum has shifted to the affected side which will itself be contracted and motionless. Since the lung is not ventilated breath sounds are absent. The percussion note is dull and vocal fremitus cannot be detected. This occurrence must be dealt with at once if complications are to be avoided. The treatment is to remove the obstruction and for this bronchoscopy may be required. It may be possible to expel the obstruction by simple percussion of the affected side of the chest. The patient is laid on his side with the healthy lung lowermost; the palm of the hand is placed over the collapsed lung in the mid-axillary line and percussed firmly with the other hand. This manoeuvre is often successful if applied early enough. If successful, the offending plug of mucus is shifted down towards the carina and is coughed up by the patient; this is followed by coughing up secretions which have collected or been retained beyond the site of obstruction. If relief of the obstruction is not prompt, mucus may be sucked out into the peripheral parts of the lung beyond the access of any bronchoscope. Once lodged there it may remain until it has become liquefied. By this time irreparable damage may have been done by local inflammatory reaction to infective organisms in the mucus plug itself which may lead to bronchiectasis. Pain and the dangerous effects of a very low intrathoracic pressure may need to be relieved by the induction of an artificial pneumothorax of sufficient volume to restore the pressure to normal; this procedure has the added advantage of forestalling further aspiration of the offending mucus plug.

Diagnosis of acute massive collapse is not usually very difficult. The chief differential diagnosis is from *massive pulmonary embolism* from which is readily distinguished by the shift in the mediastinum and the absence of air entry. In those far more frequent instances where the collapse does not involve the whole lung the diagnosis may be much more difficult. Here the signs may be confused with those of *pleurisy and effusion* and fibrosis of the lung, or of *pneumothorax* on the opposite side. Moreover, the collapse may be symptomless and be discovered only after inspection of a radiograph. A careful history will always reveal some cause for collapse; it may be obvious, as in the case of a patient recovering from deep anaesthesia who has been producing much sputum before operation or during the anaesthesia. On the other hand, the cause may be traced to a quite trifling cold in the head followed a few days later by pneumonia—due in this case to infection following aspiration of infected mucus from the upper respiratory tract during sleep. Final diagnosis is established by means of radiography which permits exact localization and may reveal the cause.

To the anaesthetist pulmonary collapse is important because it can be a complication of any operation. Every effort should be made to avoid it. Excessive sedation of patients before operation with drugs which often depress the excitability of the cough reflex should be avoided especially in debilitated patients and in those who are already suffering from suppurative diseases of the lungs such as chronic bronchitis or bronchiectasis. A course of breathing exercises and instruction in the methods of postural drainage are both valuable preventive measures. Smoking should be discouraged and the sputum sterilized by the administration of an effective combination of antibiotics before operation.

Treatment of this condition, once it has arisen, consists in removal of the cause as indicated previously. Should re-expansion not occur at once with clearing of the X-ray picture, and this is often the case, vigorous treatment should be given to counteract the pneumonia which will almost certainly ensue. Two million units of procaine penicillin should be given daily by injection combined with either one of the sulphonamide drugs in full dosage, or with tetracycline 0.5 g. a day by mouth; this should be continued for about a week at least and until the clinical and X-ray findings are restored to normal. The principal complication of pulmonary collapse followed by pneumonia is bronchiectasis; this is so serious a disease that the most energetic treatment is always advisable. Fortunately, antibiotic therapy has made this less to be feared than formerly.

Pneumothorax.

This is the condition in which the parietal and visceral pleura become separated by air. This may gain access from without, as in penetrating wounds of the chest or as the results of operations on the thorax. Occasionally, it can arise as a complication of upper abdominal operations which have involved tissues near the diaphragm. More usually

the air is admitted through a wound in the visceral pleura through which air can pass from the lung itself. Such wounds are really tears in the pleura which occur at the site of previous damage to it caused by inflammatory disease immediately below, which has healed with scarring. Another common cause is the rupture of an emphysematous bulla, but the commonest cause is a complication of active pulmonary tuberculosis. In the latter case the pleural space is often infected by tubercle bacilli which rapidly produce a serous effusion and sometimes a tuberculous pyopneumothorax; superadded pyogenic infection may further complicate the picture. Puncture of the lung by a needle during liver biopsy is not unknown and the same accident may occur during stellate ganglion block.

Signs and symptoms. These depend upon the rapidity of collapse of the lung and upon its extent. Often the pleural opening rapidly seals itself and only a small amount of air gains access to the pleural space. In less fortunate cases, the lung may collapse completely and with startling rapidity, the symptoms being correspondingly more dramatic. Occasionally the opening in the pleura may be valvular in type and thus permit the pressure within the pleura to rise above the atmospheric level. The symptoms are accordingly variable. A small symptomless pneumothorax may be revealed only by X-ray examination. On the other hand, the patient may be able to give a history of the sudden onset of severe tearing pain in the chest or upper abdomen. Severe "shock" quickly ensues and the patient becomes pale and cyanosed with increasing shortness of breath as the intrapleural pressure rises higher and higher. He becomes greatly distressed and feels as if he were being suffocated, which indeed is the case. If the air ceases to escape into the pleura the pain gradually subsides in the course of a few hours or days and the chest wall and diaphragm on the affected side are displaced to minimize the increased pressure. If air continues to accumulate heart failure soon supervenes, the patient dying from acute anoxia and circulatory collapse. In the early stages there is no pyrexia but the heart rate is high. There may be some cough which is short and dry; occasionally there may be a streak or two of blood in the sputum which comes from the torn lung tissue. The affected hemithorax moves less than the other during breathing and vocal fremitus may be absent or indistinct. Breath sounds become very weak and may be absent though occasionally sounds are conducted from the opposite side; the bronchial element of the breath sounds on the sound side may become accentuated if this lung is partially collapsed as well. When the mediastinum is mobile, it may be displaced towards the sound side, pulling the trachea with it and causing displacement of the apex beat. Vocal resonance is diminished. The clinical diagnosis may be difficult, even in complete pneumothorax; confusion with myocardial infarction or perforation of a peptic ulcer may arise as well as with massive collapse of the other lung. Careful physical examination should resolve these difficulties easily enough and a radiograph will put the matter beyond doubt. Large emphysematous bullae are occasionally confused with a pneumothorax. The prognosis is usually good with the simple pneumothorax when the hole in the lung becomes sealed. When it occurs in patients with widespread lung disease, however, the immediate outcome really depends upon the remaining respiratory function. If this is already severely impaired even a small pneumothorax may prove fatal. In the absence of this complication the air in the pleural space is completely absorbed in the course of the next two to six weeks and no harm ensues.

Treatment. The immediate treatment depends upon the severity of the symptoms. In the majority of cases seen by the anaesthetist the patient will already be in bed; if he is not, this is the first step. Careful observation is required for the next 24 hours in order to ascertain whether the pneumothorax is increasing. Half-hourly pulse and respiration charts should be kept from the start. If necessary morphine may be administered to relieve pain but this should be avoided in patients who have a seriously depleted respiratory reserve; cyanosis will direct attention to this and if it is present oxygen should be administered. In such cases alveolar ventilation is probably inadequate (and this should be confirmed by measurement of the arterial CO_2 tension) and if this is seriously impaired, more vigorous treatment of the condition may be indicated. In most instances nothing more need be done. In the case of recurrent spontaneous pneumothorax a decision will have to be taken as to whether to forestall further recurrence by the introduction of a sterile irritant into the pleural

cavity. Various solutions have been recommended for this purpose; one very good one is a 1 per cent solution of camphor in arachis oil. Others use solutions of silver nitrate and advise that it should be painted on to the surface of the visceral pleura in the place where the air leak is; this involves thoracoscopy and is probably not so frequently done now as formerly. One great advantage of using camphor in oil is that subsequent pain is not great as it often is where silver nitrate is used. If the pneumothorax is more than minimal the intrapleural pressure should be measured as soon as possible; when this is oscillating about atmospheric pressure, sufficient air should be removed from pleural space to leave the mean pressure about 1 cm of water below atmospheric pressure. Should the opening in the lung be valvular it may be necessary to insert a needle and leave it connected to an underwater drain so as to prevent a tension pneumothorax from developing. Coughing should then be repressed so as to give the pleural opening a chance to become permanently sealed. Morphine or codein or one of the newer anti-tussive drugs may be given for this. A sterile pleural effusion is a frequent occurrence as a complication of spontaneous pneumothorax; here the underlying cause is almost certainly tuberculosis and the treatment must then be directed towards the cure of this disease. If the effusion is not so large as to be a cause of respiratory embarrassment it should be left alone; it will usually subside within a few weeks. Further treatment then becomes a matter for the expert. The same remark applies to secondary infection of the effusion, with the attendant possibility of the formation of an empyaema. *Haemothorax* may sometimes occur as a complication of spontaneous pneumothorax and results from the rupture of vascular pleural adhesions. Persistently raised heart rate, in spite of adequate treatment of the pneumothorax combined with the other signs of internal bleeding, gives a reliable indication of this occurrence. Treatment involves removal of the blood, possibly replacing it with air but this, too, should be left to the expert.

Chronic bronchitis.

The reason for mentioning this disease and describing certain aspects of it in detail are that it has some features of considerable interest to the anaesthetist and others which are of great practical importance. Its frequency alone is such that the anaesthetist inevitably deals with many chronic bronchitic patients in the course of his work. This fact arises from the prevalence of two of the most important aetiological factors, namely smoking and atmospheric pollution. The disease can affect children as well as adults of all social groups but is most commonly met with amongst adults who seek advice when the symptoms become obtrusive; this may be years after their first appearance. It is true that a large number of these patients will give a history of cough which may extend backwards to childhood, and may in some examples have originated in an acute attack of bronchitis associated with whooping cough or measles. Occasionally, the start of the symptoms is dated to exposure to poison gas in the first world war. Exposure to similar noxious vapours in other circumstances is uncommon.

Symptoms and signs. The important symptom of this disease is persistent cough with the production of sputum. At first this may not be troublesome; it may even pass unnoticed for years. Eventually the patient's friends start drawing his attention to it or he falls victim to an acute attack of bronchitis and calls his doctor to see him. Up to this time the sputum has been mucoid and small in amount; a teaspoonful is coughed up in the morning (as often as not while smoking the first cigarette of the day). This increases in amount as the disease progress until several ounces a day may be expectorated, sometimes with considerable effort. Shortness of breath arises from the gradual onset of emphysematous changes in the lung; the appearance of this symptom is stealthy as a rule, but its appearance may be greatly accelerated by recurrent attacks of acute infective bronchitis. A single severe pulmonary infection of this sort may be sufficient to make the patient a respiratory cripple. Thereafter the sputum usually becomes mucopurulent, not constantly but more and more frequently with intermissions during which the sputum reverts to a mucoid appearance. Evidence has been advanced by May (1954) that the important infecting organism is the *Haemophilus Influenzae;* this is always found in purulent sputa together with other pyogenic organisms of which the most frequent is the pneumococcus.

The breathlessness arises from a combination of plugging of the smallest divisions of the bron-

chial tree by mucous followed by destruction of their walls and subsequent distortion. This greatly increases the resistance to the passage of air through these tubes rendering the ventilation of the alveoli distal to the lesions inadequate. When the disease is sufficiently widespread the effort of sucking air into the lungs and of forcing it out again becomes obvious to the patient. The obstruction is greater during expiration because at this time all the air passages are diminished in calibre. During forced expiration damaged bronchioles may become completely occluded thus trapping air in the alveoli behind them. As this process continues, destruction of alveolar walls may occur also, so that the total surface of the alveoli becomes smaller.

A chronic bronchitic patient whose disease is well established and is already complicated by the presence of emphysema presents a typical clinical picture which is familiar enough. He sits in the consulting room obviously short of breath, often expelling each breath through pursed up lips; speech may be punctuated with gasps for breath. Cyanosis is the rule but superficial examination may suggest that arterial oxygen saturation is lower than is really the case. This is because the peripheral capillaries, which are responsible for the hue of the skin, may contain blood which is much more unsaturated than the arterial blood on account of slow blood flow in the part observed. It is essential to inspect warm mucous membranes in order to arrive at a true estimate of the severity of this important sign. No excuse need be offered for mentioning this again. Inspection may reveal the existence of the barrel-chest deformity though this is by no means always the case even in the severely afflicted. Respiratory movements may be small in amplitude, indeed the chest wall may hardly move at all, and the accessory muscles may be seen contracting violently with each gasping inspiration. Abdominal movements may be exaggerated and the whole physical process of ventilation indicates the expenditure of considerable effort. The apex beat of the heart may not be visible or palpable through the chest wall, but pulsation of the hypochondrium may betray the vigorous heart action which is often found. Percussion of the chest may not contribute anything to the examination of the patient though diminution of the areas of cardiac and liver dullness is often found when the lungs are greatly distended. Hyperresonance of the chest to percussion is often described as a sign; this is difficult to be sure about unless it is very gross. With the stethoscope it is seldom possible to make out alveolar sounds because of the loud rhonchi and coarse râles which obscure them. On the other hand one of the most sinister stethoscopic signs is almost complete silence. This occurs sometimes with advanced disease as a result of intense bronchospasm; these patients are very ill indeed; the same observation is more commonly met with in status asthmaticus. In earlier, less severe examples, loud rhonchi are heard which are predominantly expiratory; often they are surprisingly less obvious when heard by the stethoscope than the noisy breathing of the patient would lead one to expect. It has been suggested that this is because the site of the obstruction—giving rise to the noise at the mouth of the patient—is in the trachea rather than in the finer subdivisions of the bronchial tree (Dekker and Groen, 1957). The heart sounds may be difficult to hear; accentuation of the pulmonary element of the second sound is sometimes heard or it may be split, indicating that pulmonary hypertension is present.

The course of the disease may be complicated by the occurrence of repeated attacks of pneumonia which may be patchily distributed or have a well defined segmental or lobar distribution. Every attack carries with it the risk of further permanent damage to the lung and must be treated promptly. Progressive respiratory failure is the usual fate of the chronic bronchitic patient who develops emphysema and this often ends in congestive heart failure. Death may be postponed by recurrent periods with rest in bed and the appropriate treatment of heart failure; to the doctor, this treatment may seem officious, and the patients themselves are seldom really comfortable when life consists of a long drawn-out battle to breathe.

Treatment. Prevention is the best method of treatment. In most cases this is not possible but it is nearly always possible to prevent the advance of the disease and the onset of emphysematous changes in the lungs when the patient is seen early enough. At the stage when the sputum is mucoid it is always wise to give up smoking. This measure alone will often be sufficient and is effective in a remarkably short space of time. Sputum ceases to be produced, cough becomes less and less frequent

and breathlessness, if it was noticeable, becomes less obvious. Recurrent respiratory infections should all be treated by rest in bed supported by efficient antibiotic treatment. Penicillin in full dosage combined with some other drug, such as a sulphonamide will deal with most of these occurrences. Tetracycline is, however, the best drug to use since it will remove *H. Influenzae* from the sputum for a time at least. The reappearance of this organism in the sputum is usually the herald of a further recurrence. The next most important element in the treatment is directed to maintaining patency of the bronchioles. Bronchodilator drugs should be administered as a routine to any patient with signs of bronchiolar obstruction; the relief they give is sometimes very great but more often it is less effective than this. Perhaps better results are obtained when these drugs are exhibited in the form of a mist from a hand nebulizer; in this way the drug exerts its local effect without having to gain access to the bloodstream first; it is thus possible to give a lower total dose and side effects are less pronounced. Solutions of ephedrine or isoprenaline in a concentration of 1 per cent are usually effective and adrenaline may be given in this way too. These solutions should not be prescribed in large quantities at a time because they do not keep well. They should be renewed every month. Some of the failures to respond to adrenaline, in particular, have been traced to the use of old stock solutions.

RESPIRATORY FAILURE

By this term is meant the failure of the lungs adequately to arterialize the mixed venous blood. One cause of this must be mentioned here for the sake of completeness. It occurs in a group of diseases of the lungs which produce an 'alveolar-capillary block' (Riley 1951). In this condition oxygen is prevented from gaining access to the blood in the alveolar capillaries by thickening of the alveolar walls themselves or by accumulation of exudates within the alveoli. The treatment is directed to the causal disease and the administration of oxygen is essential. Carbon dioxide retention does not occur in these diseases. In all other forms of respiratory failure the condition of anoxaemia is accompanied by CO_2 retention. It is useful to consider the special case of emphysema because it is in this disease that chronic respiratory failure is seen. Owing to the uneven matching of alveolar ventilation and blood flow, the mixed arterial blood contains varying amounts of blood which has flowed through territories of the lung where ventilation is very poor; such blood therefore is deficient in oxygen and has not fully shed its load of CO_2. As this state of affairs continues the respiratory mechanism ceases to compensate for the rise by hyperventilation of the remaining healthy parts of the lung; CO_2 retention becomes established and continues steadily to increase until it may reach extreme degrees. For example, there are many patients who continue to lead useful lives whose arterial blood contains CO_2 at a partial pressure of 60 mm Hg in place of the normal 40. The only method of treating this state of affairs is to improve alveolar ventilation by the appropriate amount; unfortunately this has so far defied the efforts of physicians. A surgical approach to the problem consists in reducing the dead space ventilation by instituting a permanent tracheostomy. Although this idea is theoretically a good one it is not too satisfactory in practice because of the risks to which it exposes the patient. Moreover, there is often difficulty in maintaining the patency of the tracheal stoma. The greatest misfortune that can befall the severely emphysematous patient with chronic suboxygenation and CO_2 retention is the occurrence of a widespread pulmonary infection. This at once precipitates him into acute asphyxia which can only be relieved by the administration of oxygen, and it must be so relieved, or else he will die. Unhappily the very anoxia from which he is suffering is providing a powerful stimulus to breathing and its rectification abolishes that stimulus. Breathing then becomes so shallow and ineffective that he ceases to ventilate sufficiently to get rid of CO_2 which he is producing. Carbon dioxide retention therefore increases in severity and may reach levels at which the patient becomes narcotized by his own CO_2. Carbon dioxide narcosis must always be anticipated in such patients when they are receiving oxygen therapy. The proper way to deal with this event is to attack the respiratory infection as effectively as possible. It is absolutely vital to keep the patient's own resistance as high as possible, so that oxygenation must be maintained in spite of the respiratory depression. Should this become extreme and he becomes deeply comatose, artificial ventilation

through a tracheostomy with frequent aspiration of retained secretions should be resorted to without delay. At the same time spasmolytic drugs, such as isoprenaline, should be given as a mist intermittently with oxygen, and respiratory stimulants, such as nikethamide, may be given by intramuscular injection every two hours. Recently it has been suggested that the depressing effect of pure oxygen might be avoided by giving concentrations of oxygen in the inspired gas in the region of 30 per cent, that is in sufficient amount to correct the suboxygenation partly without completely abolishing the anoxic drive. This can only be accurately controlled where there are facilities for rapid gas analysis; it should be remembered that the majority of patients suffering from CO_2 narcosis recover when their chest infection subsides even when they are given pure oxygen. One of the best ways of administering it is by nursing the patient in an oxygen tent. Alternatively, it may be given through a pair of Tudor Edwards' spectacles or mixed with air through one of the Venturi tube devices recently recommended by Campbell (1960). This last device fitted to a light transparent mask is an extremely handy and effective one and is well tolerated by patients.

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ENDOCRINE DISORDERS IN ANAESTHESIA

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DISORDERS of the pituitary, pancreas, parathyroid, thyroid and adrenal glands may adversely influence the response of a patient to anaesthesia or surgery and so are of particular concern to the anaesthetist. In this connexion diseases of the ovaries or testes are of no importance and will not be discussed further.

Patients with endocrine disease may appear to the anaesthetist in one of two quite distinct roles. The first is the patient with some known endocrine disorder that is to be treated surgically. This seldom presents a problem because in most instances the physician and surgeon will have done everything possible to reduce the risks of operation. The second is the patient who presents with a non-endocrine disease requiring surgical intervention, but who in addition is suffering from an unrecognized glandular disturbance. From the anaesthetist's point of view this second type of patient is the most important if disaster is to be avoided, and often it is the astute anaesthetist who first suspects the additional non-surgical condition during his pre-operative assessment of the patient as a whole.

UNRECOGNIZED ENDOCRINOPATHY

In patients with undetected endocrine disease symptoms are often wholely absent or minimal. Once the condition has been suspected and appropriate direct questions asked, the patient may admit to having noticed some abnormality, but usually the onset of the glandular deficiency is so insidious that the patient is unaware that anything is amiss, and his relatives fail to notice the striking changes that are occurring in his appearance and behaviour. Particularly is this the case in hypothyroidism, whether due to primary thyroid disease or secondary to pituitary insufficiency. In this type of patient routine history taking or the asking of direct questions often fails to raise any suspicion of endocrine disease. True if a sufficiently large number of symptoms are asked about, hints of some endocrine disorder may arise, but the number of questions that would have to be asked to uncover the many possible syndromes makes this impracticable.

There are, however, four questions which should always be asked of any patient about to undergo an operation. *Weight-loss* is common to many non-endocrine surgical conditions, but if unexplainable on this basis or disproportionate to the major disease, may indicate diabetes mellitus, hyperthyroidism or adrenal insufficiency. *Polyuria* or polydypsia, which may be equated by the patient with insomnia, may indicate diabetes mellitus, diabetes insipidus or hyperparathyroidism. Rarely *menstrual disorders* may indicate some major endocrine disorder; amenorrhoea being a symptom of pituitary failure, hyperthyroidism or adrenal insufficiency, and menorrhagia of hypothyroidism.

The fourth question or series of questions, which must never be omitted, is to discover if the patient has ever had systemic treatment with corticosteroid drugs. As regrettably few still have little idea of the nature of any tablets received in the past, it may be necessary to enquire if the patient has suffered from any of the diseases commonly treated with cortisone or its analogues—asthma, hay fever, rheumatoid arthritis or urticaria. The importance of this, to be discussed more fully later, lies in the adrenal suppression induced by steroid therapy—a suppression which may last for two years after treatment has finished.

Physical examination is often helpful in uncovering unsuspected endocrine disease, and only a few seconds are required to detect the diagnostic features. To the eye that sees, hypothyroidism is immediately obvious, yet this is one of the most commonly missed diagnoses, even in its grossest form, and recently two such patients were submitted to operation in a teaching hospital—fortunately without disaster. The pinkness of the cheeks against the yellow pallor of the rest of the

face and the cold dryness of the skin on the limbs are characteristic features of hypothyroidism whether due to primary thyroid disease or secondary to pituitary insufficiency. Facial, axillary and pubic hair is often sparse and may be wholly absent in long-standing pituitary deficiency in which gonadal hypofunction is added ot the hypothyroidism. The rubicund moon facies of Cushing's syndrome is also characteristic but may be confused with non-endocrine obesity: in the former condition there is a curious disparity between the fatness of the trunk and upper part of the limbs and the slender delicate structure of the wrists, ankles, hands and feet; whereas in the latter the obesity is uniformly distributed. Acromegaly is unlikely to be overlooked. The pigmentation of Addison's disease may be attributed to racial origin but has an uneven distribution being most marked on areas subjected to pressure—the knees, elbows, waist where belt or skirt band press, and shoulders and thorax where brassiere straps chafe. Pigmentation is usually marked in the inside of the mouth, on the gums below the incisor teeth and in old scars. A low blood pressure is not common except during an adrenal crisis. The neck should always be inspected and palpated: even moderate enlargement of the thyroid as in a simple colloid goitre may add to the difficulties of intubation. Glycosuria is best detected with Clinistix which is speedy, sensitive and specific for glucose—being uninfluenced by any other reducing substance. The presence of acetone in the urine indicates either severe diabetes mellitus or malnutrition—both of a degree which should be corrected before operation.

In general the greater the degree of endocrine dysfunction, the more obvious are the physical signs and the greater the operative risk. In adrenal suppression secondary to steroid therapy physical signs are wholly absent, and the one most often found in diabetes insipidus or hyperparathyroidism is mild dehydration.

RECOGNIZED ENDOCRINOPATHY

Once an endocrine abnormality has been diagnosed or is suspected, the management of the patient will depend on the urgency of the surgical condition. Whenever possible the diagnosis of the endocrinopathy should be confirmed by appropriate investigation, and corrective treatment instituted. In many conditions, however, response to treatment is slow, surgical intervention cannot be delayed and the pre-operative state of the patient is less than ideal. Nonetheless the risk is immeasurably reduced by the anaesthetist's awareness of the endocrine abnormality and his readiness to counter any difficulties that may arise during or after the operation.

Thyroid disease.

The presence of a goitre or any irregularity in the thyroid substance should immediately give rise to the question: "Is this patient thyrotoxic?" Minor degrees do not materially add to the risk of surgery on other parts of the body, but patients who are more severely affected may have auricular fibrillation, right-sided heart failure or liver damage, which may to some extent offset the increased rate of metabolism of sedatives and hypnotics usually found in hyperthyroidism. Unfortunately there is no treatment that will reduce the activity of the thyroid within a matter of hours. Reserpine in large doses (5 mg intramuscularly thrice daily) will allay the tachycardia, restlessness, tremor and other overt manifestations usually attributed to the peripheral action of excess thyroxine. Potassium iodide, 5 drops of a saturated solution thrice daily, will inhibit the release of thyroxine from the gland but an appreciable effect is not achieved until after 4–5 days, and the maximum response occurs after 10–21 days. Chlorpromazine, hypothermia and intravenous hydrocortisone (200–300 mg) comprise the treatment of choice for a thyroid crisis following thyroidectomy in an inadequately prepared patient with known hyperthyroidism, or in a patient with a nodular goitre erroneously thought to be non-toxic.

Hypothyroidism seriously delays the metabolism of drugs and renders the patient unduly sensitive to them. The operative risk is further increased because of secondary changes in the pituitary and adrenal glands which impair the normal responses to stress. In general the greater the degree of hypothyroidism and the longer its duration, the greater is the operative risk. There are, however, curious individual differences in response.

Two men of the same age and, judging from subsequent basal metabolic rate estimations, suf-

fering from an equal degree of hypothyroidism, were operated upon before recognition of their endocrine disease. One had a herniorraphy and responded normally to his pre-operative medication and anaesthesia. The other showed undue sensitivity to the pre-operative medication and developed severe hypotension during transfemoral aortography which necessitated abandoning the investigation.

Myxoedema retards the responsiveness of the adrenal glands to ACTH and there is similar evidence that the pituitary in the myxoedematous subject is less responsive to whatever stimuli increase its activity. Thus the development of severe hypotension in these patients is thought to be due to adrenal insufficiency; they should be treated with intravenous hydrocortisone hemisuccinate or prednisone-21-phosphate.

If time permits an attempt may be made to correct the hypothyroidism before operation. Tri-iodothyronine acts in a few hours but must be used cautiously in a patient over the age of 50 and particularly in any patient who has a history of angina pectoris or evidence of atherosclerosis. Under-treatment is safer than over-treatment, and the dosage of tri-iodothyronine should not exceed 10–20 μg, 12-hourly.

Adrenocortical disease.

Adrenocortical insufficiency may develop post-operatively after adrenalectomy for Cushing's syndrome (or for the control of carcinomatous metastases), in patients with Addison's disease, and in those having corticosteroid treatment or who have stopped such treatment weeks or months before surgery. This last group contains the largest proportion of patients who are exposed to the greatest risk: pre-operatively they have no abnormal physical signs and the possibility of adrenal suppression being present as a consequence of previous corticosteroid therapy can only be uncovered by direct questioning. Too many patients are unaware of the nature of therapy received in the past and the physician who prescribes corticosteroids has a moral obligation to explain to the patient the physiological consequences and to provide him with a written note of warning and instruction.

All synthetic analogues of cortisone and hydrocortisone cause pituitary inhibition and secondary adrenal suppression. The degree of suppression is more related to the duration of corticosteroid treatment than to dosage, and there are wide, and as yet unexplained individual variations. Any patient currently having steroid therapy is particularly at risk, but the risk persists for weeks or months after stopping treatment even when the dosage is gradually reduced. Irreversible shock has been described after minor surgical procedures in patients who have stopped steroid therapy for as long as 24 months, but have sufficient adrenocortical function to allow a life free of symptoms under more ordinary circumstances.

Attempts to prevent adrenal suppression by giving adrenocorticotrophin concurrently with steroid therapy have not been wholly successful in maintaining the normal responsiveness of the pituitary-adrenal axis to stress. Injections of corticotrophin induce histological changes in the pituitary similar to those found after prolonged administration of corticosteroids, and collapse has occurred during surgery in patients who have had prolonged treatment with ACTH alone. Thus in the practical management of corticosteroid treated patients, administration of ACTH cannot be relied upon to prevent operative complications, and time seldom allows the application of somewhat complicated tests to assess pituitary-adrenal responsiveness. The wisest course is to give prophylactic treatment to any patient currently having steroid treatment or who has had prolonged treatment during the previous two years. These two types of patient constitute an absolute indication for special pre- and postoperative care. Patients whose treatment ended more than two years previously may face an additional operative risk, but usually special pre-operative preparation is unnecessary provided appropriate measures are available and are implemented immediately if collapse occurs.

Corticosteroids are rapidly absorbed from a normally functioning gastro-intestinal tract. An appreciable rise in the plasma steroid concentration occurs within half an hour of administering the tablets; it reaches a maximum in 1–1½ hours although the metabolic consequences of the increased steroid level may not be manifest for another 3–4 hours. In contrast to many substances corticosteroids are not well absorbed after intramuscular injection; for speed of action this route of administration is much inferior to oral or

intravenous therapy, and plays little part in the emergency treatment of adrenal insufficiency. In prophylaxis, however, intramuscular cortisone provides a useful depot from which absorption will continue for many hours. For intravenous use hydrocortisone hemisuccinate or prednisone-21-phosphate, which are both water soluble, are used in the knowledge that a major operation in a normal individual will call forth the secretion of about 400 mg hydrocortisone in 24 hours from healthy adrenal glands (equivalent to about 100 mg prednisone).

For patients receiving steroid treatment and those who have had corticosteriods during the preceding two years, the amount of additional steroid required depends on the severity of the operation and the degree of adrenal suppression. Both factors are difficult to assess and as too little supplementation may lead to disaster, larger rather than lesser amounts should be used. The same principles should guide the preparation of a known or suspected Addisonian patient. It is now appreciated that patients with partial adrenal insufficiency may lead a normal life for many years and yet succumb suddenly and unexpectedly to some quite trivial operation. The disease is so slowly progressive that the patient may fail to report any symptoms, or only admit to increased fatigue or minor abdominal discomfort and looseness of the bowels on direct questioning. Often pigmentation of minor degree is the sole abnormal finding. Excretion of hydrocortisone and its metabolites in the urine as determined by the currently available routine laboratory methods may not be abnormal and adrenal insufficiency may only be revealed by finding diminished responsiveness to stimulation with ACTH. Whenever the suspicion is raised it is wiser to provide prophylactic coverage.

Forty-eight, twenty-four and again six hours before operation the patient is given 100–200 mg cortisone acetate intramuscularly. If currently taking steroids, the patient continues with his usual dose in addition. At the completion of the operation another 100–150 mg cortisone is given by injection. Subsequent management will depend on whether the patient is vomiting or not. If possible cortisone is given orally in a dose of 50 mg three or four times on the first postoperative days. Thereafter the dosage is reduced gradually over the next week and stopped after 10–14 days in those not having current steroid therapy, or reduced to the pre-operative maintenance dose in those who are. If the patient is vomiting, intramuscular cortisone is used—100–150 mg on the first and second post-operative days and 75–100 mg on the third. Thereafter oral treatment should be used but at all times hydrocortisone hemisuccinate (or prednisone-21-phosphate) must be readily available for intravenous administration if the blood pressure falls or unexplained pyrexia develops.

The same pre-operative treatment should be used for patients with Cushing's syndrome undergoing adrenalectomy. If the cause of the condition is an adenoma or carcinoma, atrophy of the contralateral adrenal will have occurred, and removal of the tumour will be followed by adrenocortical insufficiency. If the cause is bilateral adrenal hyperplasia, total adrenalectomy will certainly lead to adrenal deficiency, and subtotal adrenalectomy will often have the same effect until the adrenal remnant begins to function again.

For patients who have stopped steroid treatment more than two years previously, it is usually safe to omit prophylactic measures provided the treatment (outlined later) is immediately available if collapse develops during or after operation. The same steps should be taken if collapse occurs in patients already having supportive steroid therapy. If the fall in blood pressure is detected early and appropriate action is taken at once, the patient will respond promptly. It is often asked whether this form of treatment should be applied when hypotension occurs unexpectedly and unexplainably during an operation on a patient supposed to have normal adrenal function. There is no doubt that if the hypotension is due to adrenocortical insufficiency it will respond to intravenous steroid therapy given promptly, but that this form of treatment is without apparent benefit in collapse due to other causes. Since there may be no means of determining the aetiology of the hypotension it seems prudent to give steroids intravenously and observe the therapeutic response. More often than not some non-endocrine cause will later become apparent but no harm will follow the administration of intravenous hydrocortisone.

As soon as collapse occurs, 100 mg hydrocortisone (as the sodium succinate salt), or 20 mg of

prednisone or prednisolone (as the 21-phosphate) is injected intravenously. An intravenous infusion of glucose-saline is set up and 500 ml containing a further 100 mg hydrocortisone given over the next twelve hours. During the first 24 hours 400 mg hydrocortisone may have to be infused in 1–1½ l. of fluid. On some occasions 2–4 mg noradrenaline have been added to the infusion bottle; it is doubtful whether this is necessary when adequate amounts of hydrocortisone are used. Furthermore, because of the undesirable local vasoconstrictor effects of noradrenaline, "Aramine" is preferable if the blood pressure does not rise in response to corticosteroid treatment.

Phaeochromocytoma.

The anaesthetist plays an important part in the surgical treatment of phaeochromocytoma. Preoperatively the hypertension can be controlled by oral administration of phenoxybenzamine, and the resulting persistent sympathetic blockade will allow considerable improvement in the patient's cardiovascular system. The dosage required usually varies between 20 and 200 mg daily and is adjusted to produce the desired reduction in blood pressure. During the operation, handling of the tumour may induce extreme elevation of the blood pressure and fatal cardiac arrhythmias. Anaesthetic agents, such as cyclopropane, which may produce cardiac arrhythmias should be avoided, and sudden rises in blood pressure checked with intravenous phentolamine in doses of 0.1 mg/kilo body weight. A precipitous fall in blood pressure after removal of the tumour should be countered by the intravenous infusion of noradrenaline, which is best administered through a polythene catheter introduced into the superior vena cava (to avoid local ischaemic effects in the arm).

Diabetes insipidus and hyperparathyroidism.

The pre-operative preparation of patients with diabetes insipidus, or hyperparathyroidism, is directed towards correcting dehydration, and maintaining the fluid balance postoperatively at a time when the patient cannot drink but the urinary loss of water continues unabated. Pitressin tannate in oil, 2–5 units subcutaneously, will stem the diuresis in diabetes insipidus, but in hyperparathyroidism intravenous infusion of isotonic glucose solution may be required to make good the fluid losses.

Diabetes mellitus.

Diabetes is perhaps the endocrine disease most often encountered by the anaesthetist and the one which poses the most difficult problems. The situation is straightforward if the patient is known to have diabetes which is under good control—his treatment is left unchanged until the day of operation and provided there are no renal complications, the operative risks are not substantially different from those of a non-diabetic patient. Other patients, although known to have diabetes, may become inadequately controlled as a result of the surgical condition requiring treatment, or because they have deliberately but wrongly reduced the dosage of insulin when anorexia or vomiting diminished their food intake. Others are not discovered to have an abnormality of carbohydrate metabolism until their urine is tested on admission to hospital. It is essential in these patients with glycosuria to assess the severity of the diabetes. First it is necessary to confirm that the reducing substance detected with Benedict's solution, or Clinitest tablets, is truly glucose, and this is done with a specific glucose-oxidase method such as Clinistix. Although the severity of the diabetes is to some extent reflected by the degree of glycosuria, ketonuria is the most important simple guide. This must be tested for with Acetest tablets, or ferric chloride solution, but the latter may give a positive result if the patient has been taking salicylates or *p*-aminosalicylic acid. The presence of ketonuria calls for immediate emergency treatment and the operation must be delayed at all costs for at least 6 – 12 hours. More accurate assessment must be obtained in patients who are uncontrolled or in whom the disease was previously unrecognized, by determining the blood sugar level and the degree of acidosis as shown by the CO_2 combining power. A blood sugar level of 250–300 mg per cent in the absence of ketonuria calls for some adjustment of the insulin dosage in a patient known to have diabetes, and the institution of insulin therapy in a young person who gives a history of thirst, polyuria and weight loss; in a middle-aged or older overweight person, who on direct questioning denies any diabetic symptoms, no special preoperative measures are necessary. When the blood sugar level exceeds 300 mg per cent and there is concomitant acidosis as shown by ketonuria

and a lowering of the CO_2 combining power, insulin therapy and correction of dehydration are essential pre-operative steps.

Pre-operative preparation. The treatment outlined here is designed for those patients who require insulin therapy but are not in diabetic coma or precoma. The objectives are to eradicate any ketosis; control the blood sugar level and avoid hypoglycaemic reactions. Most patients will have marked glycosuria and some ketonuria but the CO_2 combining power will not be reduced below 20 m.equiv/l. Food is given 6-hourly in four equal amounts providing 100 g. carbohydrate, 100 g. protein and about 50 g. fat per day. Whether this food should be solid or liquid will depend on the patient's surgical condition. Insulin is given six-hourly before each meal. Soluble insulin is used because its duration of action is comparatively brief. If the patient had been taking insulin, the first dose should be one-third the total previous daily dose irrespective of the type of insulin used before. If the patient has never required insulin in the past, the initial dose should be 12–16 units. Thereafter the amount of insulin given is determined by the amount of sugar excreted in the previous six hours. If the urine is red or orange, 20 units are injected; if green or yellow, 10 units.

Day of operation. It is important to avoid hypoglycaemia on the day of operation because unconsciousness may erroneously be attributed to the anaesthesia. Provided ketosis is avoided it does not matter if the blood sugar level is undesirably high and it is safer to have glycosuria rather than no sugar at all in the urine. The principle is to feed the patient every six hours and give insulin before each feed.

Usually parenteral feeding will be necessary on the day of operation because the stomach should be empty when anaesthesia is induced and the patient may vomit postoperatively. A simple and satisfactory regimen is to give 500–750 ml of 5 per cent glucose during each 6-hour period. This can be given by mouth or intravenously according to the patient's condition, and during every third period the glucose should be dissolved in physiological saline rather than water. Soluble insulin is used throughout and administered subcutaneously. If the patient has been satisfactorily stabilized pre-operatively the first period should be preceded by an amount of insulin which is one-quarter of his previous total daily requirements. Thereafter the amount of insulin must be judged from the amount of glucose excreted in the urine.

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RENAL AND ELECTROLYTE DISORDERS

BY

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INTRODUCTION

THE fluid and electrolyte composition of the body may be disturbed because of an excessive strain on the normal regulating mechanisms, or because the regulating mechanisms are so impaired that they cannot deal with normal metabolism. Physiological stresses, such as infection, vomiting or surgery, accelerate protein breakdown and result in greater amounts of nitrogenous products, acid metabolites such as phosphates and potassium being presented to the kidneys for excretion. When there is dehydration or low blood pressure, kidney function is directly impaired and the resulting disturbance is correspondingly greater. For the present purpose, therefore, it seems artificial to divide the clinical problems into renal or non-renal groups.

The physician's appraisal of fluid and electrolyte disturbances is based initially on the following points: (1) the history of fluid losses and intake; (2) the state of hydration; (3) evidence of diminished blood volume; (4) the presence of certain clinical features of uraemia; (5) the level of respiration; and (6) the chemical composition of the plasma. The various disturbances, either clinical or biochemical which may be detected, will be discussed in turn without reference to the underlying disease. In practice, several of these disturbances may co-exist, but it is simpler to think of them as separate problems in order to understand them.

DEHYDRATION

This term is usually used when there is a deficiency of extracellular fluid volume. If the deficiency is of water alone, it will be spread through both extracellular (25 per cent of body weight) and intracellular (35 per cent of body weight) compartments; whereas, if it is of salt and water, the deficit may be confined to the extracellular compartment. The physical signs by which it is recognized—dry tongue, dry skin with loss of turgor and elasticity, soft eyeballs, low blood pressure with small pulse volume, collapsed veins and oliguria—are all nonspecific, and it can be difficult to assess the degree of dehydration in a chronically ill patient who has lost weight. Many of the above signs may be found in the presence of oedema, but unless there is a local cause for it, oedema excludes dehydration. Dehydration is harmful chiefly because of its effect upon the circulation through important organs, such as the kidneys, and so in its assessment most weight should be placed upon the clinical signs of low blood volume. The simplest laboratory aids in the estimation of blood volume are the haematocrit, total protein, and serum sodium levels, but it is obvious that these are affected by the illness and not just the state of hydration. Direct measurements of blood volume are not very difficult, but most clinicians feel that they are rarely necessary for therapeutic purposes. If dehydration is responsible for oliguria, the urine has a high specific gravity and high urea concentration. Dehydration which persists for some time may result in acute renal failure due to renal damage, and then the urine becomes dilute. The simplest way of deciding between oliguria due to renal damage of the tubular necrosis type and simple dehydration is to determine the urine urea concentration. If this is below 1 g./100 ml kidney damage must be held responsible (it is usually about twice the blood level in acute renal failure), whereas if it is over 2 g./100 ml the oliguria is likely to be explained by pre-renal factors (Taylor, 1957). The blood urea level may be equally high in both conditions.

Management.

The correction of dehydration is simple in principle—it is by the administration of fluid by mouth or by vein. The two problems are: how much fluid and of what kind? The amount of fluid required has to be guessed, but there are several general principles for guidance. Initial assessment of the history and serum sodium should indicate whether the deficiency is of water alone or salt and water. As a general principle, about 5 per cent reduction in a compartment is necessary to produce physical signs, and about 20 per cent reduction is near the limits that can be tolerated. The ranges of fluid deficit usually experienced are, therefore, about 800 ml to 3.5 l. of extracellular fluid, or, if the deficit is pure water, from 2 l. to 8 l. An estimate of the severity of the dehydration enables a figure between the two extremes to be chosen. In practice, it is convenient to estimate first the amount of pure water deficit on the basis of the elevation of serum sodium and to administer this amount as 5 per cent glucose. When the serum sodium has been corrected, or if it is initially normal or lower, it may be assumed that the remaining deficit is of solute solution, and this can be administered as saline. Thus, a moderately severely dehydrated man might present a serum sodium of 150 m.equiv/l. (7 per cent above normal). One would, therefore, give for an average sized man 7 per cent of 40 l.; i.e., nearly 3 l. of 5 per cent glucose. If at the end of this time he still appeared dehydrated, further fluid would consist of saline solution. If the serum sodium level were initially normal, and one arbitrarily decided on a figure of 10 per cent as an estimate of the reduction in his extracellular volume, one would give for the same man 10 per cent of 17.5 l. of saline. If serum sodium figures are not readily available, the decision about whether to use saline on the one hand, or glucose solution on the other hand, rests on the history. Losses from the gastro-intestinal tract may be regarded as essentially isotonic for electrolytes, whereas water deprivation is primarily a water deficit.

The term "saline solution" has been used so far for replacement of extracellular fluid, but a more physiological solution, such as Lactated Ringer's Solution, is better if kidney function is impaired. The decision that dehydration has been adequately corrected depends primarily on the overall clinical impression, but two points should be emphasized: (1) the appearance of sacral oedema is a signal to stop, and (2) persisting oliguria should only be taken as evidence of persisting dehydration if the urine is tested and found to have a high specific gravity, or a urea concentration over 2 g./100 ml.

SIGNIFICANCE OF BLOOD UREA LEVEL

Renal failure results in the clinical features and blood chemistry changes of uraemia. A fairly accurate guide to both the diagnosis and the severity of the failure is given by the blood urea nitrogen (BUN) level. The normal fasting level of BUN is 10–15 mg/100 ml. (Blood urea level is a little over twice the BUN level.) After a protein meal, the BUN rises and then slowly falls, so that even the fasting level reflects the protein content of the diet. In renal failure the BUN may rise to approximately 100 mg/100 ml with very little clinical evidence of uraemia. There are, of course, wide individual variations, but as a general rule the symptoms of uraemia develop as the BUN reaches a value of 70–100 mg/100 ml, and increases in severity as the BUN rises further. They may be severe enough to result in death any time after the BUN has exceeded 200 mg/100 ml. It is popularly believed that urea itself is nontoxic, and that the urea level is merely a signpost to the degree of retention of other toxic products of metabolism. This is not entirely true, for experimental work with dogs has shown that urea alone can produce the signs of uraemia and death. Patients who have developed uraemia slowly seem able to tolerate elevated levels of BUN better than in the case of acute renal failure, but in all patients a BUN of over 200 mg/100 ml is associated with a hazard to life.

It should be remembered that the BUN reflects the balance between renal function and the body metabolism. The serum creatinine is a better guide to renal function as such, but the BUN takes into account the protein intake and any increased products of metabolism resulting from stress, and it is the BUN that should be used as a guide to clinical state. The level of urea production may rise many fold as a result of stress. Thus, in acute renal failure the daily rise in BUN in uncomplicated examples is usually less than 15

mg/100 ml, but in traumatic or infected patients, it usually exceeds 30 mg/100 ml (Parsons and McCracken, 1959). In pre-operative assessment, it must be remembered that the postoperative increase in urea production will almost certainly cause a transient rise in blood urea level, even if periods of hypotension or dehydration are avoided. There is, of course, no absolute level of BUN at which one can say surgery is contra-indicated, but the operative hazard will increase as the BUN level rises. Where the operation is designed to relieve acute urinary obstruction, as for example, a nephrostomy in a patient with acutely obstructed ureters, the BUN falls immediately after successful operation, and even patients with very high initial levels of BUN may tolerate the operation well. After chronic prostatic obstruction, on the other hand, some time elapses before improvement in renal function is marked, and after prostatectomy a rise in BUN level is to be expected. It is often taught that since the main source of urea in the body is the liver, severe liver disease may be associated with a normal BUN, even in the presence of renal failure. This is a fallacy. In the various types of acute hepato-renal syndrome, the BUN rises in the same way as in pure renal failure, and it is quite common to have elevated BUN levels developing in patients with terminal cirrhosis, even though liver coma may be present (Papper et al., 1959).

SIGNIFICANCE OF THE SERUM POTASSIUM LEVEL

The plasma or serum level of potassium (normally, 3.5–4.5 m.equiv/l.) is often elevated in renal failure. The elevation may result from either inability of the kidney to excrete adequate amounts of potassium, or from acidosis disturbing the distribution of potassium across cell membranes. Similarly depression of the serum potassium level may result from either potassium depletion or from alkalosis. The main toxic action of disturbed levels of serum potassium is upon neuromuscular activity. It occurs in cardiac, voluntary and unstriped muscle, but the dangerous effects are those upon the heart and on the muscles of respiration. High levels of serum potassium depress the conduction through the heart and result in characteristic e.c.g. changes. When death occurs from hyperkalaemia, it is usually as a result of sudden stopping of the heart in diastole without necessarily any previous clinical evidence of disturbed heart action, although arrhythmias may occur. For this reason, high levels of potassium should be regarded as very hazardous. The danger to the heart is not dependent upon the absolute level of serum potassium only, but is increased by low levels of serum sodium and decreased by high levels of serum calcium, so that many clinicians advise using the e.c.g. as a guide, in addition to the serum potassium level. However, a high level of potassium should never be regarded with complacency merely because the e.c.g. changes are minor. As a general guide, patients are unlikely to die from hyperkalaemia when the level is below 7 m.equiv/l., and none of them can tolerate a level of 10 m.equiv/l. It should be remembered that anoxia, and perhaps any stress, results in increased liberation of potassium from body cells.

Low levels of serum potassium may result from alkalosis, or from potassium depletion. Because of the other factors involved, it is not possible to estimate the amount of depletion by the serum level, or to assume that no depletion is present if the serum level is normal. Moore (1959) believes that some loss of acid in addition to potassium deficit is nearly always present in hypokalaemia in surgical patients. An acidotic patient with a normal serum level may have a masked potassium deficiency, and on correction of the acidosis there may be a sudden drop to subnormal serum levels. There is considerable variation among different patients in the serum level at which muscular paralysis develops. As a general rule, serious symptoms are not likely to develop with a potassium level above 2.5 m.equiv/l., although subjective weakness, loss of tendon reflexes and e.c.g. changes may occur earlier (McCardle, 1959). The biggest danger from hypokalaemia is that of a sudden respiratory paralysis; usually paralysis of the limb muscles occurs first, but this cannot be relied upon. The effect of hypokalaemia on the heart is to increase muscular irritability, and cardiac arrest in systole may be produced. An important aspect of hypokalaemia is that it increases the toxic effects of digitalis. In patients who are digitalized, the development of hypokalaemia may be associated with signs of digitalis toxicity, and in

patients with hypokalaemia, digitalis must be used cautiously.

Management of hyperkalaemia.

High levels of serum potassium may represent maldistribution of potassium, rather than excess potassium in the body. Correction of any co-existing acidosis will often correct the hyperkalaemia. High levels of serum potassium may be reduced by removing potassium from the body. This removal may be achieved by haemodialysis, where techniques for this are available, or by ion exchange resins. Resonium A, for instance, given as a retention enema, 45 g. in 100 ml water, may be repeated at 12-hourly intervals and may be expected to reduce the serum level by amounts of the order of 1 m.equiv/l. per enema. As a temporary expedient, potassium may be removed from the circulation by giving glucose and insulin (e.g. 500 ml of 10 g./100 ml glucose with 15 units of soluble insulin in the bottle), but as the glucose is later metabolized, the potassium is released into the circulation again. In addition to these measures, some protection against a high level of potassium is afforded by the infusion of a calcium salt or by raising the level of serum sodium. Thus the most rapid measure to protect against hyperkalaemia in an emergency is the intravenous administration of sodium lactate. Where the amount of sodium that can be given safely is limited, it can be followed by an infusion of glucose and insulin. As a general rule the emergency measures should be followed up by more lasting ones, such as resin therapy or haemodialysis.

Management of hypokalaemia.

Low serum potassium levels may be associated with alkalosis, and here correction of the alkalosis will cause a rise in serum potassium. To the extent that the low serum level is due to potassium depletion, it may be corrected by the administration of potassium. The potassium may be given as potassium chloride pills (1 g.=13 m.equiv), or the standard potassium citrate mixture used for urinary infections (1 g.=10 m.equiv). If the potassium has to be given parenterally, it may be given as a potassium-containing solution such as Darrow's (contains 35 m.equiv/l.), or it may be added as potassium chloride concentrate to any convenient fluid. The principles of parenteral administration are that the solution given should not be stronger than 40 m.equiv/l. (it causes pain along the vein), and it should not be given faster than 40 m.equiv/l./hour. The amount of potassium required in patients with depletion varies from 100 to 1000 m.equiv, but normally replacement at the rate of 150 m.equiv per day is adequate, with daily checks upon the serum level. There is no real danger of over-administration of potassium, provided that it is not given too quickly and that there is a reasonable urine output.

SIGNIFICANCE OF THE SERUM SODIUM LEVEL

In renal disease sodium may be retained, or it may be lost. Where sodium is lost excessively, it usually results in diminished extracellular fluid volume and hypovolaemia at the same time as low plasma levels of sodium, and this state of dehydration is recognizable clinically. When the level of sodium in the plasma falls, there is necessarily a drop in the osmotic pressure of the extracellular fluid, and a resulting shift of water into the cells. If this occurs when the overall fluid depletion is slight, or has been corrected by water or glucose solution the excess water in the cells constitutes water intoxication. In practice, most patients with sodium depletion have a combination of low extracellular fluid volume and low plasma sodium; i.e., a combination of extracellular dehydration and intracellular water intoxication. This is the typical picture seen in Addison's disease.

Low plasma sodium also occurs in the absence of sodium depletion. This is most commonly seen in chronic illnesses in the presence of oedema, and the low sodium level largely represents a dilution phenomenon. The low osmotic pressure in the extracellular fluid would be expected to result in a water shift, and thus water intoxication of the body cells, but there is often associated with this picture a depletion of intracellular ions (chiefly potassium), and the water content of the cells may be relatively normal. There is usually a partial replacement of intracellular potassium by sodium. The importance of recognizing this state is that attempts to correct the plasma sodium level by administration of sodium chloride are dangerous; they will result in a further increase in extracellular fluid volume,

and perhaps pulmonary oedema, at the same time as the plasma sodium rises. Cort and Mathews (1954) have shown that administration of potassium alone, to restock the cells, results in a return of the plasma sodium towards normal.

In practice, symptoms of hyponatraemia are very variable, and some patients may tolerate a sodium level of 110 m.equiv/l., without any apparent disturbance, while others have mental confusion at 120 m.equiv/l. In general, however, levels of sodium above 120 m.equiv/l do not cause symptoms unless they are associated with water intoxication.

Management of hyponatraemia.

Correction of hyponatraemia by giving sodium will always result in an increase in extracellular fluid volume, because of the shift of water distribution. Thus correction of hyponatraemia by giving sodium should be undertaken only when there is evidence of sodium depletion from the body and when some expansion in extracellular volume would be either desirable or, at least, harmless. To raise the serum sodium from, say, 120 m.equiv/l. to 140 m.equiv/l. enough sodium has to be given to raise the concentration 20 m.equiv/l. in the extracellular fluid, and also to raise it 140 m.equiv/l. in the water that shifts out of the cells. For this reason it is necessary to use the total body water, rather than extracellular fluid volume only, when calculating the rise in serum sodium to be expected from a given amount of sodium (Welt, 1959). For a 70-kg man, this would require about 800 m.equiv of sodium which would result in the expansion of the extracellular fluid by approximately 3.5 l. It is obvious that many patients would not be able to tolerate this. If there were no sodium depletion initially, this procedure might result in thirst, and if water were allowed freely there might be a redilution of the sodium level and still further oedema. One gramme sodium chloride contains 17 m.equiv sodium, and 100 ml of 3 per cent sodium chloride contains 51 m.equiv.

ACIDOSIS

The pH of blood (normally 7.4) may fall in the metabolic acidosis of renal failure to levels as low as 7.1. This acidosis is harmful in many ways, and is probably responsible for much of the nausea and vomiting that may occur. The clinician often expects to be able to recognize acidosis by the hyperventilation that is usually associated, but this hyperventilation is not a reliable guide. The respiratory centre seems to become adapted to pH changes so that in chronic acidosis the ventilation may be normal. It is seldom that blood pH values are available, and usually one relies on the plasma CO_2 combining power as an indicator of acidosis. In metabolic acidosis the CO_2 combining power falls from its normal level of 25–30 m.equiv/l. to as low as 5–10 m.equiv/l. In acute acidosis, acidotic breathing is usually evident when the CO_2 value falls below about 15 m.equiv/l. Hyperventilation is a compensatory mechanism and, for a given level of CO_2 combining power, the actual pH of the blood will be less acid in the presence of deep breathing and more acid if breathing is shallow. The pH depends upon the fraction $\frac{H_2CO_3}{BHCO_3}$ and CO_2 combining power is a measure of the denominator, so that the shallower the breathing, the higher will be the numerator and the greater the acidosis. This is of obvious importance to anaesthetists who have the respiration under their control and who are normally used to assessing the adequacy of respiration in terms of oxygen. When metabolic acidosis is corrected rapidly, hyperventilation usually continues for some hours and may, therefore, cause a transient respiratory alkalosis (Parsons and McCracken, 1959), so that in this context the degree of hyperventilation may be misleading as an index of the degree of acidosis.

Management of acidosis.

The treatment of respiratory acidosis is the direct province of the anaesthetist, and only metabolic acidosis will be considered here. Where the acidosis is due to ketone bodies, in diabetic coma, the acid radicals can be metabolized under the stimulus of insulin therapy. Acidosis due to fixed acids can be most completely rectified by haemodialysis, because this is the only therapeutic means whereby they can be removed rather than just neutralized. However, in practice the acidosis is usually treated by neutralization. When sodium bicarbonate is tolerated by mouth, this method can be employed. Intravenous therapy may consist of sodium lactate or bicarbonate. The limiting

factor to this method of treatment is essentially the amount of sodium the kidneys can excrete. When the kidneys can excrete large amounts of sodium, acidosis can be readily corrected by this means, but in cases of oliguria it may not be feasible to correct the acidosis without causing pulmonary oedema. The amount of sodium lactate or bicarbonate required can be estimated by the level of CO_2 combining power in the plasma. If, for example, the level is 12 m.equiv/l., the deficit in bicarbonate is 15 m.equiv/l., or, in a 70-kg man, a total of $15 \times 18 = 210$ m.equiv in the extracellular fluid. This calculation provides a marked underestimate, because some of the bicarbonate given (or formed from lactate) will be used to neutralize the acidosis present in the cells, and some will be converted to carbonic acid when fixed acids are released from other buffers as the pH returns toward normal. The calculation is worth making, however, as an initial guide for therapy. One litre of isotonic sodium lactate (M/6 lactate) contains 167 m.equiv of lactate.

Alkalosis and its management.

Respiratory alkalosis will not be considered here. Metabolic alkalosis may occur from excessive ingestion of alkali, as happens sometimes in peptic ulcer patients from excessive vomiting of acid gastric juices; it occurs in potassium depletion and after long-term use of diuretic drugs. As a general rule, the underlying condition must be treated in order to improve the alkalosis. In potassium deficiency, for instance, administration of potassium is all that is required. In alkalosis due to alkali ingestion, stopping the intake is usually adequate because the normal metabolism of the body produces an excess of acid products. In the alkalosis of repeated vomiting, where chlorides have been lost from the body, treatment consists of replacing the chloride as ammonium chloride or potassium chloride, depending upon the serum potassium level. The number of milliequivalents of chloride required can be roughly estimated from the serum chloride level by multiplying the deficit by the extracellular volume. The alkalosis which sometimes follows long continued use of mercurial or other diuretics may be partly due to chloride losses, and partly potassium deficiency. It is probably best treated by giving both potassium and ammonium chloride by mouth, perhaps 3 g. daily of each.

CLINICAL ASSESSMENT OF URAEMIA

When a patient has clinical evidence of uraemia, he is seriously ill. The clinical features that are perhaps of most importance in assessing the gravity of the situation are (1) central nervous system irritability, (2) pulmonary oedema, and (3) a falling blood pressure. In the first group are included muscular twitches, which often herald the onset of convulsions, and these may result in death. The blood pressure is usually maintained fairly well in uraemia, but it often falls steadily during the last day or two of life.

Other features of importance in relation to preoperative assessment are:

Haemorrhage.

Patients with uraemia have a tendency to excessive oozing of blood, spontaneously from mucous membranes, and from surgical wounds. This oozing may persist for days. Routine tests of bleeding and clotting mechanisms are usually normal, and this bleeding tendency must be assessed clinically from inspection of the mouth, nose and cut-down sites.

Anaemia.

The anaemia of chronic renal failure, in the absence of significant haemorrhage, is roughly proportional to the elevation of BUN. In acute renal failure there may be no anaemia at first.

Glucose tolerance.

In renal failure glucose tolerance is impaired. In ordinary medical management this is of little consequence, but during administration of intravenous glucose the blood sugar may be quite high. This might be of some significance in potentiating the effect of barbiturate anaesthesia (Lamson et al., 1951).

Infections.

Patients in renal failure are more susceptible than normal people to bacterial infections (e.g., staphylococcal). Since they are more susceptible to antibiotic resistant infections, as well as the sensitive ones, they should receive extra aseptic care rather than prophylactic antibiotics. Elective operations might well be preceded by bacteriological study of the faeces and pharynx to detect

antibiotic resistant staphylococci, and in this way if antibiotics become necessary, those likely to result in staphylococcal enteritis could be avoided.

Management of renal failure.

This is not the place for a treatise on the management of renal failure. There are certain aspects of its management, however, that are important in connection with pre- and postoperative care. Preoperative preparation with a protein-free or low protein regimen, containing adequate glucose, will reduce to a minimum the protein catabolism, and will, therefore, place the patient in the best position to withstand the increase in catabolism which will result from surgery. Any disturbance in acid-base balance or electrolyte pattern should be corrected pre-operatively, if possible, on general principles. The low serum calcium which is often present in patients with renal failure does not usually cause any difficulties, but if tetany is present, or muscular twitching, an infusion of a calcium salt may be given with transient benefit. In anaemic patients, blood transfusions are often indicated pre-operatively, but the increased breakdown of the older red cells present in the transfusion will aggravate any existing hyperkalaemia and acidosis. Even in the absence of obvious imbalances, there is an increased operative mortality in patients with high blood urea levels. Although we do not know that this increased mortality is due to biochemical disturbances which can be corrected by haemodialysis, there is a good case for preparing selected patients for surgery by dialysis a day or two beforehand.

ANURIA

This represents the most complete form of renal failure. Its management consists essentially of complete protein and potassium restriction, the giving of at least 100 g. glucose daily, and a very careful regulation of water balance. More detailed descriptions of its therapy may be consulted (Merrill, 1955; Parsons and McCracken, 1959). Although the anaesthetist may be called upon in the treatment of a patient with anuria, this is more likely to follow surgical treatment than to precede it. The greatest service the anaesthetist can render in this regard is to minimize the risk of anuria developing. Anuria seems to be most commonly a result of renal ischaemia, and is usually associated with shock but not always. The conditions prevailing in periods of trauma or surgery seem to predispose to renal shutdown, and an effort should be made to prevent undue diminution in blood volume being allowed to continue for any length of time. If periods of hypotension have occurred during operation, the ward staff supervising the patient should be told, so that the diagnosis of renal shutdown is not delayed when it arises.

TETANY AND HYPOCALCAEMIA

Hypocalcaemia is a complication of parathyroidectomy, either accidental, associated with thyroidectomy, or deliberately for removal of an adenoma. These patients are given calcium gluconate intravenously in an emergency, and calciferol or A.T.10 by mouth. It takes many hours for the calcium level to fall after parathyroid removal, and the resultant tetany will be discovered in the early postoperative period rather than in the operating theatre. Since laryngeal spasm may be the presenting symptom, the anaesthetist may be the first person called upon to make the diagnosis of hypocalcaemia.

Hypocalcaemia is also seen characteristically in renal failure, where it is a response to the elevation in serum phosphorus. If tetany occurs, it may be controlled by calcium gluconate infusion, but the effect quickly disappears after the infusion is stopped. There is also a defect in absorption of calcium in patients with renal failure, and this can be rectified by large doses of calciferol.

OEDEMA

There is a tendency in individual patients to accumulate oedema either completely in the peripheral connective tissues or to distribute it partly in the lungs. It is, of course, in the latter type of patient that the presence of oedema causes most concern to anaesthetists. Pulmonary oedema is not confined to patients with left ventricular failure, but is found in a variety of pathological conditions, and it is probably desirable to reduce the degree of oedema in any patient.

The treatment of oedema by the standard means of salt restriction and mercurial injections of chlorothiazide-type tablets is usually effective. Resistant cases of oedema may be of several

kinds, but a few general principles may be mentioned. (1) In hypochloraemic alkalosis, administration of ammonium chloride or potassium chloride may restore responsiveness to mercury. (2) In hyponatraemia, water restriction may be cautiously tried. If this results in oliguria it should be stopped. There is very frequently associated potassium depletion, even though the serum potassium level is normal, and the hyponatraemia may improve if potassium chloride is given. This is long-term therapy, however, and not suitable for pre-operative correction. (3) Where the serum electrolytes are not grossly abnormal, and there is inadequate response to mercury or chlorothiazide, the simultaneous use of an aldosterone inhibitor (Aldactone) may be very satisfactory. The rationale and technique have been described by Shaldon et al. (1960).

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THE ANAESTHETIST'S ASSESSMENT

Some Factors to be Considered at a Pre-operative Visit

BY

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THE anaesthetist—asked to give his opinion about the fitness of a patient for anaesthesia—must consider many factors besides the physical state.

The usual form of the question "is the patient fit for an anaesthetic?", is misleading in its emphasis; and the nature of the problem is clarified if the anaesthetist rephrases it to, "should I give an anaesthetic to an individual for a particular operation under the circumstances prevailing?" This means that a moral judgment is being made and its implications must be faced, for the anaesthetist must always give his opinion as a physician, not as a technician.

PHILOSOPHICAL CONSIDERATIONS

The anaesthetist's conscience may be involved in many ways.

(1) The type of operation, e.g., sterilization or termination of pregnancy, even if not directly prohibited by his religious beliefs, will always require careful scrutiny of all the circumstances.

(2) His appraisement of his own capabilities can also affect the issue. A junior anaesthetist may justifiably hesitate before taking on a very ill patient. Even his senior might be equally reluctant to undertake a highly specialized procedure such as conducting an extra-corporeal circulation without sufficient experience.

(3) His assessment of the surgeon's capabilities. This may sound presumptuous, but it is generally agreed that speed and gentleness of the surgery may still be of importance in some operations. Consider, for example, the position in a busy unit, where a consultant surgeon frequently and rightly, delegates operative care to his junior colleagues. It may be that he is not fully informed of the condition of some of the patients and the anaesthetist may feel that a particular patient is' ill, and that the operation should be performed by the chief. He should have no hesitation in making this suggestion. A situation also may arise where the anaesthetist is so out of harmony with the surgeon, not only over questions of personality, but also about the treatment of the patient, that the latter's welfare may suffer, particularly if he is very ill. It is surely preferable that the anaesthetist should not, if possible, give an anaesthetic in these circumstances.

(4) Predominantly out of common humanity, but partly also on medico-legal grounds, the anaesthetist should satisfy himself that the patient knows that he is to have an operation and understands the nature of it.

(5) If the anaesthetic or operation involves an experimental procedure or a clinical trial, the anaesthetist should satisfy himself that it is ethically justifiable. This problem has been conveniently reviewed in a monograph by Beecher (1959).

(6) For many emergency operations, no matter how desperate the risk, the moral problem is simpler. Something has to be done even if all the desirable facilities are not immediately available. Many other operations are labelled "emergencies" only for temporal or administrative convenience, and the anaesthetist must not allow himself to be hustled into giving an anaesthetic to a patient without adequate psychological or pharmacological preparation, or resuscitation. On the other hand, much mental and financial distress may be caused by an unreasonable postponement of operation. The interests of the patient are paramount, and a nice judgment is often required to balance the possible ill effects of waiting, against those of curtailing the usual pre-operative preparation. The risks of vomiting and regurgitation in this context are discussed later.

(7) For elective operations for conditions not endangering life, the highest standard of pre-operative fitness and preparation are essential, and not even the slightest extra risk should be taken. There are many conditions which, though not endangering life, may yet cause an elderly patient such distress that he is prepared to accept an additional risk. The benefits of operations such as prostatectomy in men and the repair of prolapse in women should not be denied on the grounds of age alone.

(8) The anaesthetist must have a clear idea of his own philosophy about the justification for operation in patients requiring "heroic" or even hopeless surgery. The surgeon must not have to feel that he is continually having to persuade the anaesthetist to continue against his better judgment, and the latter must always remember that the patient has asked for help for "something to be done". For the patient to awaken to find an abdominal mass which he hoped would have been removed, is a truly demoralizing experience. On the other hand, the anaesthetist must learn when a tactful suggestion that the patient

has "had enough" will synchronize with the surgeon's wishes in a difficult situation. No rules can be laid down and the patient's clinical condition is not necessarily the main consideration in the decision to curtail an operation. Even cardiac arrest and apparent death are seldom indications for abandoning all operative intervention.

If the fundamental principle of pre-operative preparation—relief or alleviation of intercurrent pathology—is observed and surgery and anaesthesia are of a high standard, then the overall result—in terms of physical and mental health—depends largely on the success of the operation in relieving the patient's symptoms (Simpson and Smith, 1960).

With these philosophical considerations in mind and a knowledge of the immediate circumstances, the anaesthetist may then proceed to appraise the patient further.

THE PRE-OPERATIVE VISIT

As this is made not only to assess the physical state of the patient but also to reassure him, to prescribe premedication and to make certain that he is recognized in the anaesthetic room, the time available is inevitably short. The anaesthetist must therefore concentrate his attention on selected aspects of the problem which are either neglected or not adequately stressed in routine examinations. Hospital documents, if available, should be scrutinized first. Not infrequently they reveal evidence of drug sensitivity, previous cortisone treatment, or refer to the complications of previous operations or of intercurrent diseases, which may have been overlooked at the present admission. On the strength of the information given in the notes it may be decided that only a superficial inspection of the patient is required. On the other hand they may indicate that more thorough investigation is necessary.

As in all consultations, the history is of paramount importance, and an inquiry about previous anaesthetic experience may lead to unexpected information about untoward sequelae of anaesthesia and operation, and may bring to light incidents relevant to the condition for which the patient has been admitted, which were not elicited by earlier interviewers.

Complications of previous operations may be of prognostic value. For example, a history of pneumonia after an operation on a limb is unusual, and suggests that this was an aspiration pneumonia, and that the patient perhaps regurgitates easily, and may have a hiatus hernia. A history of the symptoms of this abnormality—heartburn, retrosternal pain and regurgitation on lying down—should then be sought, particularly in the very obese. These patients must be regarded as likely to regurgitate under anaesthesia (Dinnick, 1960) and suitable precautions taken (such as passing a stomach tube before operation or inducing anaesthesia in the reverse Trendelenburg position).

Knowledge of the postoperative history of a patient with chronic bronchitis may help in assessing his chances of withstanding further surgery. If, for example, he underwent cholecystectomy without a postoperative pneumonia, the outlook is favourable for a further laparotomy. If, however, he had been gravely ill with a pulmonary infection after all his previous operations, the prognosis is correspondingly poor for further surgery.

A history of collapse under a previous anaesthetic should not automatically lead to the assumption that the anaesthetic had been badly given. Collapse may follow a period of airway obstruction and the patient may have a build of jaw or tongue which predisposes to this, or else may have a paralysis of the recurrent laryngeal nerve.

The suspicion of prolonged recovery often proves groundless on closer questioning, but a history that a patient has been put on a respirator, gives a clear warning for caution with relaxant drugs.

"Sensitivity" to anaesthetic drugs always merits careful questioning, and although it usually appears likely that a simpler explanation would fit the facts, it may be wise to omit the suspected drug from the anaesthetic sequence.

At his pre-operative visit, the anaesthetist must also elicit information, usually by direct questioning, about the patient's exercise tolerance, the presence of chest pain, cough, the amount of sputum, and whether he has had a recent cold. The history of functional impairment is usually of greater significance to the anaesthetist than the physical examination.

With increasing experience of patients with severe cardiac disease it may be said that, in general, the cardiac contra-indications to in-patient anaesthesia are very few, and of these,

recent coronary infarction is the most important. It would seem reasonable to wait 3 or 4 weeks until the myocardial scar is firm, even though not entirely healed.

The fact that there is, however, no reliable method of forecasting when coronary infarction will occur, tends to reduce the value of many cardiological assessments in the restricted sense of fitness for operation. The decision whether to operate or not is therefore usually made on surgical or other grounds.

The importance of making a detailed assessment of "bronchitis" is discussed later, as is the associated problem of "emphysema". The anaesthetist must also inquire about other current medical treatment, drug sensitivity and steroid therapy.

While this will provide a minimum of basic information which it is essential to know in every patient, many of the points elicited may require further elaboration. Whether this is the case depends partly on the findings of the clinical examination, and partly on the nature of the proposed operation as will be discussed later.

BASIC CLINICAL EXAMINATION

As the anaesthetist seldom has much time to spare in the wards, he must develop a shortened form of clinical examination to assist him in choosing the anaesthetic technique, and to enable him to screen patients rapidly, so that significant diseases are not overlooked. Skilled history taking obviates unnecessary examination. It takes but a moment to inspect the face for colour, the mouth for loose or diseased teeth, the tongue and skin for dehydration, and the shape of the jaw and neck for possible difficulty with intubation.

Auscultation of the chest will reveal irregularities of cardiac rhythm and possibly some murmurs, as well as râles and—the more likely portents of technical difficulties—rhonchi. It should not take long to diagnose congestive heart failure or gross lung pathology. Arm veins may be inspected as the blood pressure is taken. Finally, skin sepsis should be noted as well as arthritis and deformities which would interfere with the proper positioning of the patient on the table.

It is suggested that such a basic examination, which need seldom take more than a few minutes, will give sufficient information for the anaesthetist to plan his anaesthetic procedure and detect any pathology which will need further investigation. His findings, however, are only of limited value unless they are considered in relation to the following questions:

(1) Does anaesthesia by itself involve an immediate risk to life?

(2) Are there likely to be late complications due solely to the anaesthetic?

(3) Is there intercurrent disease which will affect the management of the anaesthetic?

(4) Will the proposed operation be of immediate benefit to the patient?

(5) Will the operation temporarily or permanently impair the patient's condition, particularly his pulmonary or cardiovascular reserves?

(6) Will the operative procedure adversely affect the course of intercurrent disease?

(7) Are late surgical complications likely to occur, and if so, is the patient able to withstand them?

(8) Is the patient healthy and the operation a minor one?

Although to a certain extent arbitrary, these questions must be considered in relation to every proposed anaesthetic, for the answers given will affect the emphasis required in the pre-operative questioning and examination, as well as the overall opinion about "fitness" for operation. For this reason the questions will be used as a basis for the classification of certain pathological states which merit more detailed discussion.

(1) *Anaesthesia as an immediate risk to life.*

In the past, the anoxia associated with the struggling and breath holding which so frequently accompanied an open ether induction, was often sufficient to overtax an already poor circulation with fatal consequences. This particular hazard can now usually be circumvented, however; all general anaesthetics may cause some degree of peripheral vasodilatation and fall of blood pressure, even if given slowly and skilfully and with adequate pre-oxygenation. Nevertheless, today it is true to say that, excluding the moribund, few patients will fail to survive a well chosen and skilfully administered general anaesthetic. Exceptions occur in the presence of upper respiratory obstruction, pulmonary disease or circulatory collapse when the anaesthetic procedure may gravely interfere with the patient's oxygen supply, primarily to the myocardium. Serious cerebral

anoxia is usually, but not invariably, secondary to anoxic circulatory failure.

Upper respiratory obstruction. Oedema of the glottis from radiotherapy, carcinoma of Ludwig's angina, which is often associated with trismus, are all well known; they are easily recognizable when the patient is using his accessory muscles of respiration. With lesser degrees of obsruction, as with thyroid enlargement causing tracheal compression, the anaesthetist can obtain useful information by asking the patient to inspire as rapidly as possible. If this is markedly prolonged and accompanied by a guttural hiss and the accessory muscles can be seen in action, induction and intubation will be difficult and may end in disaster; tracheostomy should be seriously considered. Conversely, if there is only slight noise and no prolongation of inspiration, anaesthesia will probably be uneventful.

The use of the accessory muscles of respiration is, however, not the only warning of potential respiratory obstruction under anaesthesia; indeed, laryngeal spasm may complicate any anaesthesia and obstruction of the pharyngeal airway may be expected in many patients whose airway is perfectly clear when they are conscious. Examples of this may occur in patients with acromegaly, arthrodesis of the temporo-mandibular joint, macroglossia, micrognathos, ankylosing spondylitis of the cervical spine, and patients who have had a resection of mandible or a fractured jaw. Little harm will result if the anaesthetist proceeds with caution and inserts a tube through the nose; but a thoughtless induction with a large dose of relaxant may sometimes rapidly lead to a dangerous situation. Rarer causes of airway obstruction include upper mediastinal masses and paralysis of the recurrent laryngeal nerve; suspicion should be aroused by recent hoarseness or by a scar in the neck.

Pulmonary disease. When anaesthesia is likely to be an immediate threat to life, two possibilities are of paramount importance, firstly, the risk of drowning in pus from a lung abscess or empyaema, and secondly, and far more important, severe respiratory spasm. While the former hazard may usually be prevented by the use of blockers or posture, the latter is often intractable and is likely to be made worse by thiobarbiturates and by the irritation of the respiratory tract by some inhalational anaesthetics. Relaxant drugs are of no assistance and controlled respiration is impossible when the spasm is chronic and severe, as in status asthmaticus.

Local or regional analgesia is strongly indicated for many of these patients, and in those with very severe "emphysema" or with cor pulmonale, where bronchospasm and cyanosis persist in spite of energetic medical treatment. Indeed, it is arguable that this small group of patients is the only one in which regional analgesia is absolutely indicated on medical grounds, as distinct from the non-availability of drugs, equipment, skilled staff, or the personal choice of the patient or medical attendant.

In the absence of severe and persistent bronchospasm and excessive purulent sputum most patients with even gross pulmonary disease can usually be safely anaesthetized.

Fatal cardiovascular collapse. When this is the immediate result of anaesthesia it is usually due to overdosage. Nevertheless, it may also follow a carefully given anaesthetic in patients with gross heart disease or in patients who have had very recent myocardial infarction—within 2 weeks—or who have continuous anginal pain. The same applies to patients with constrictive pericarditis or severe aortic valve disease, in some patients with left heart failure and also in those with severe pulmonary hypertension. Further deterioration is likely in those with a blood pressure either unrecordable or not more than 50 mm Hg following severe haemorrhage.

While the possibility of lethal upper respiratory obstruction must be considered in every patient, surgery is seldom, fortunately, required for cases with gross pulmonary and cardiovascular disease.

(2) *Late complications due solely to anaesthesia.*

These may be pulmonary, toxic, neurological or the result of hypotension, and while the blame for many pathological conditions may be put on the anaesthetic, few should be due solely to this cause.

Pulmonary complications are discussed later, and are inextricably related to the site of the operation, the extent of pre-existing lung disease and to upper respiratory tract infection. The only pulmonary complications directly due to anaesthesia are those which are the result of inhalation of vomitus, which is considered elsewhere, mishaps during intubation and pulmonary collapse from the faulty management of anaesthesia.

Toxic effects on the liver are the known sequelae of certain drugs, such as chloroform or chlorpromazine, the use of which can be avoided particularly if liver damage is already suspected. Similarly, renal failure may occasionally follow anaesthesia and operation, but there is little satisfactory evidence that it is ever caused by the established anaesthetic agents.

Neurological sequelae of various regional analgesia techniques are well recognized though rare. More commonly, they result from faulty posture in the unconscious patient.

Hypotension if profound or prolonged may lead to grave sequelae in the central nervous system and occasionally in other parts of the body. Some attempt must always therefore be made to estimate the patient's ability to withstand it. This is particularly important if it is likely to occur during the proposed operation or is to be induced deliberately by ganglion blocking drugs or other means. Nevertheless, such an assessment is not easy, because it is known that prolonged and severe hypotension is frequently encountered during major surgery without any untoward complications. It would seem that this state is less harmful than has been supposed, provided there are no other causes of hypoxia, such as pulmonary disease, severe anaemia or technical faults, and, of even greater importance, provided that circulatory collapse does not occur or is allowed to remain unrecognized and untreated for more than a few seconds. This possibility is always present, even in normotensive patients, but is more liable to occur in those with hypertension, especially with advanced atherosclerosis or with poor myocardial reserve and coronary artery disease. Hypotension should not be allowed to occur in these patients or in those, usually the aged, in whom there is a history of a previous cerebral thrombosis.

Muscle pains after suxamethonium sometimes amount to much more than discomfort and may even delay a patient's discharge from hospital. It therefore seems reasonable not to use this drug simply to produce respiratory arrest in procedures such as retrograde pyelography or aortography, where postoperative pain may be suggestive of untoward complications. The author is aware of several such instances where diagnostic confusion was caused because this possibility was not considered, and on more than one occasion, a cardiological opinion was sought because of suspected coronary thrombosis.

Most of the complications already mentioned do not usually follow a competently given anaesthetic, and are more properly considered as therapeutic misadventures or errors of technique. The possibility of their occurrence, however, must always be considered by the anaesthetist during his assessment; this may influence his choice of anaesthetic technique.

(3) *Intercurrent disease affecting anaesthesia.*

The anaesthetist's assessment is concerned with the detection of these diseases, with ensuring that the patient's condition is improved as much as is practicable before operation, and with the problems of anaesthetic management caused by these disorders. A full discussion of the subject is beyond the scope of this paper although several conditions are of necessity briefly considered in other sections.

(4) *Immediate benefit from the proposed operation.*

From the anaesthetist's point of view the majority of emergency operations may be considered under this heading, although some of the more major and less urgent groups fall into the next category, i.e., where the benefit of the operation is delayed.

Patients requiring emergency operations may be either moribund, or capable of responding to resuscitation or may be otherwise fit.

Moribund patients. The term moribund applies to the state of patients with multiple pathology, advanced liver disease, uraemia, heart failure, cachexia and intractable electrolyte disturbances. It implies that medical treatment cannot further improve their condition.

Occasionally, an operation, such as opening an abscess, may be justifiable in a moribund patient, and the anaesthetist may be asked for his opinion. Some guide to the immediate outcome of anaesthesia may be obtained from a modification of the breath holding test. If a breath can be held for 5 seconds anaesthesia will probably be uneventful, but if the patient is unable to hold his breath at all the outlook may be grave, but light anaesthesia with, e.g., cyclopropane is, even then, not invariably fatal. Such conditions are, fortunately, rarely encountered.

"Normal" emergencies. Of greater importance are the frequently encountered abdominal and traumatic emergencies, where much can be done to improve the patient's condition before surgery.

Assessment is primarily concerned with (a) resuscitation and the optimum time for surgery, and (b) with the risk of regurgitation or vomiting.

Resuscitation. The necessity for adequate preoperative blood or electrolyte replacement has long been realized, and it is customary to aim at restoring the systolic blood pressure to at least 80 mm Hg before inducing anaesthesia. While this figure may be regarded as a minimum if no further loss is anticipated, it is preferable to aim at a higher level.

Of recent years there has been a greater awareness of the surprisingly large amounts of blood required after injury and of the need for electrolytes for patients with intestinal obstruction or peritonitis. Less widely appreciated perhaps, is the rapidity with which these fluids may safely be given to an exsanguinated or dehydrated patient with a healthy myocardium. If citrate intoxication is prevented by giving 1 g. of calcium gluconate to each pint of blood, and if the transfusion tubing is passed through a water bath at 37°C blood may be given as rapidly as it will pass through the intravenous needle. This is routine practice in major thoracic surgery, and is frequently equally applicable to resuscitation. Blood or fluid can be given until the peripheral veins are again full, and the capillary refilling after the release of pressure is almost instantaneous. Frequent auscultation of the lung bases will provide a check against overtransfusion.

If, in spite of energetic transfusion the blood pressure fails to rise, and if it is obvious that the patient is still bleeding, immediate operation is essential. Similarly, in some patients with volvulus or mesenteric thrombosis, there may come a time when no further improvement can be obtained by transfusion. Laparotomy should then no longer be delayed.

The full stomach. For the majority of emergency operations such heroic resuscitation of the patient is not required, but in all of them the risk of vomiting or of regurgitation must be contemplated by the anaesthetist.

There are two different liabilities to be considered; firstly, where regurgitation is likely, as with intestinal obstruction, advanced peritonitis, burst abdomen and in many obstetrical patients, particularly when labour has been prolonged; and secondly, those who have recently had a meal, although the tendency is less marked.

The problems involved in the preparation and management of the former group are now well known. (Morton and Wylie, 1951; Snow and Nunn, 1959). While a Ryle's tube cannot be relied on to empty a stomach, it does permit gastric distension to be considerably reduced, and thus materially lessens the risk of regurgitation. This risk, however, still remains, partly because the stomach may not be completely emptied, or may have refilled from retrograde peristalsis, and partly because the intra-abdominal pressure may still be high. Further precautions are therefore essential, and the technique recommended by Morton and Wylie (1951) and Snow and Nunn (1959) of inducing anaesthesia in the head-up position is gaining favour, and has much to recommend it. Two contra-indications to this technique must always be considered in patients likely to regurgitate. These are the potential difficulty of performing rapid intubation for anatomical reasons, and the patient's inability to withstand the inevitable fall in blood pressure associated with inducing anaesthesia in this position. Sufficient resuscitation with intravenous fluid and a suitable vein are essential prerequisites.

Extubation and emergence from anaesthesia are equally dangerous periods for these patients, who should always be kept head down and in the lateral position, unless it has been possible to pass a large tube under anaesthesia to empty the stomach.

All injured patients should be regarded with suspicion because gastric emptying usually ceases. The interval between the last meal and the accident—not between the meal and the induction of anaesthesia—should be considered in assessing the risk of vomiting. The risk of regurgitation is less after injuries than in the other group, as solid food, will not regurgitate, neither can it be withdrawn through even a large stomach tube. Similar precautions are therefore desirable during induction, and are essential during the recovery from anaesthesia.

While not strictly relevant to the circumstances just described, it is convenient to discuss here the common

situation where, owing to a misunderstanding or to a disruption of operating schedules, a patient has taken food or drink within the commonly accepted period of 4 hours before anaesthesia is to be induced. It is customary, and indeed widely accepted by the medical profession and by the public, that it is prudent to postpone the operation in these circumstances, and no anaesthetist who does so need fear criticism for his decision. Such a decision will inevitably cause the patient considerable mental distress.

The basis for postponing operation for this reason is therefore worthy of reconsideration, for although the basic assumption that the stomach normally empties within 4 hours is usually correct, this is not the case in very nervous individuals. Many children are nervous, and others defy instructions not to eat or drink in the pre-operative period, and indeed, not a few adults are equally remiss in this respect, and are as successful as the children in concealing their misdemeanours.

Since, therefore, "routine precautions" are not really to be trusted, and since so many patients requiring emergency operations are successfully managed by modern anaesthetic techniques, it is suggested that there is a case for these techniques to be employed for suitable patients who have inadvertently received a small meal before a scheduled operation. In further defence of this admittedly unorthodox viewpoint, it should be stressed that it is the unexpected vomit that leads to disaster; if the possibility is clearly anticipated, and the necessary precautions taken, no harm should result.

(5) *Temporary postoperative impairment of the patient's condition.*

Postoperative deterioration is usually due to the disorder from which the patient has been suffering and to the effects of surgery, or impairment of pulmonary function. (Other surgical complications will be ignored in this context.) Haemorrhage should be amenable to treatment and the anaesthetist should always make a pre-operative assessment of its probable extent, and ensure that adequate supplies of blood are available.

The risks of postoperative pulmonary complications must always be seriously considered. They may prove lethal by a direct reduction in the amount of functioning lung, especially if that is already below normal before operation. They may also strain an already damaged myocardium.

Except where due to aspiration either of vomit or of infected droplets in an upper respiratory infection, pulmonary disorders in surgical patients are usually preceded by atelectasis. This is affected by two main factors which usually operate simultaneously: (1) restriction of respiratory excursion and of the ability to cough and (2) excessive secretion of sputum.

The former is the inevitable accompaniment of all upper abdominal and thoracic operations, and to a lesser extent of those on the lower abdomen, including the repair of an inguinal hernia. Patients for operations of this kind therefore need particular attention at the pre-operative stage, particularly with regard to the excessive secretion of sputum.

Clinical examination is relatively unrewarding, and though numerous rhonchi should always arouse suspicion an inspection of the sputum pot is usually more valuable. Uncontrollable coughing is obviously of bad import.

The history, however, is most important, and an appreciable amount of time is usually required for questioning patients with "chronic bronchitis." The extent of winter disability, smoking habits, duration of exacerbation after a cold, response to previous operations and the patient's own opinion of the present state of his disability, are among the factors to be considered in deciding whether the patient's sputum production is at its minimum, or whether it can be reduced further by treatment. Elective operations on the upper abdomen and chest should always be postponed until sputum production has been reduced to a minimum by energetic postural drainage and physiotherapy—combined with the use of bronchodilator drugs and perhaps detergent aerosols.

Conversely, patients about to undergo operations on the limbs or superficial tissues need not detain the anaesthetist for long. Even those with severe bronchitis are unlikely to run into trouble after operations of this kind unless respiration is very severely depressed for much longer than is customary, and coughing is also discouraged.

The common cold in the writer's experience, always merits close attention from the anaesthetist. He should inquire into the usual course of the disorder in the patient, and further information can be obtained from the progress of the disease in contacts when there is an epidemic. As this disorder may be the precursor of further pulmonary infection, especially in elderly patients, without the added strain of anaesthesia and operation, it should never be regarded lightly. In patients with chronic bronchitis, particularly if the operation is one which predisposes to atelectasis, postponement until the infection has resolved is strongly indicated, and such a course is usually desirable

when similar operations are proposed for patients with healthy lungs. However, for other operations it is usually safe to proceed as soon as it is clear that the disease is past its worst.

"Emphysema." The term "chronic bronchitis" has been used to describe the disorder so commonly encountered in this country, but patients so affected usually also suffer from "asthma" and "emphysema". All three terms are extremely unsatisfactory, and the need is now recognized for a more precise terminology to define the many varied anatomical and functional disorders of the lung to which these names have been applied (Ciba symposium, 1959).

It is not possible to summarize the important recommendations made at this symposium, but one proposed new term, "generalized obstructive lung disease, reversible or persistent", is of particular interest to anaesthetists because it emphasizes the functional changes which occur in this group of diseases.

The simple clinical assessment of "chronic bronchitis" is reasonably satisfactory in that it provides a basis for decisions about pre-operative treatment and the optimum time for operation. It also enables most bad risk patients to be detected, but it is important for the anaesthetist to realize its limitations.

Marked impairment of pulmonary function due to obstructive lung disease is well known to be associated with severe "asthma"; and the presence of loud, persistent rhonchi will always alert the anaesthetist to the likelihood of technical difficulties and postoperative complications. What is less widely appreciated is that equally severe pulmonary dysfunction due to obstructive disease may be present when the classical signs of obstruction are not prominent.

Indeed, the anaesthetist usually only becomes aware that his patient's lungs are grossly inefficient after the anaesthetic is commenced. He may then find that the inhalational induction is markedly prolonged, or if he has used a relaxant drug, artificial ventilation is remarkably ineffective in that there is difficulty in keeping the patient oxygenated. At the end of operation spontaneous respiration may be difficult to re-establish, and a state of carbon dioxide narcosis may be recognized.

The place of the more complex pulmonary function tests in making more detailed pre-operative assessment of such patients has yet to be defined. Many of the tests lack precision (Shephard and Turner, 1959), and their time-consuming nature and often overriding claims of surgical urgency will probably preclude their widespread adoption except in special units.

The simpler single breath tests are more valuable, and merit wider use especially to determine the extent to which a patient's obstructive disease is reversible, and to measure the response to pre-operative medical treatment.

The facilities for performing these tests are not, however, widely available yet, but even without them the anaesthetist can gain valuable information on the extent of the total disability due to obstructive lung disease by a simple procedure. This is done by asking the patient to blow out a match while holding his mouth wide open. The test has still to be properly evaluated, but it would seem reasonable to regard a patient who is unable to perform this task as a very poor risk for upper abdominal surgery, and one for whom more detailed investigation is desirable, particularly of his arterial blood carbon dioxide tension (P_{CO_2}).

Measurement of the mixed venous P_{CO_2} by the rebreathing method (Campbell and Howell, 1960) is relatively simple, and will enable impending or established carbon dioxide narcosis to be diagnosed so that further treatment, for example by tracheostomy, may then be based on factual knowledge rather than on intuition.

The merits of regional analgesia for the patient with marked impairment of pulmonary function due to obstructive lung disease, should always be seriously considered. Some of the difficulties associated with controlled respiration in these patients have already been considered. There is also some evidence that the vascular changes due to sympathetic block (epidural or spinal) improve pulmonary ventilation (Bromage, 1954), but a beneficial effect, as shown by changes in the P_{O_2} and P_{CO_2} do not necessarily follow such a change.

(6) *Immediate adverse effect of anaesthesia and operation on intercurrent disease.*

Stress from a major operation may result in a flare up of a dormant infection. Tuberculosis

used to be a notable example, but this is no longer likely because adequate chemotherapy is available. However, a similar response from quiescent malignant disease is not merely a theoretical possibility, and deserves more than casual consideration. The immediate concern of the anaesthetist is to ensure than any essential maintenance drugs, normally given by mouth, are replaced by parenteral equivalents over the operative period. Steroid and the antidiabetic drugs are the most common examples.

(7) *Surgical complications.*

Although primarily a question for the surgeon, surgical complications must also be considered by the anaesthetist. Pulmonary embolism is always a hazard of surgery, and is more likely in patients who have been confined to bed before operation. Patients coming to surgery from medical wards have often been so treated; the anaesthetist should consider this possibility, and postponement to allow a more active pre-operative regimen is sometimes desirable.

The anaesthetist will also frequently discover that a surprisingly large and varied amount of antibiotic drugs have been taken by patients, and indeed, they may still be taking them unknown to the surgeon. Such information is always valuable, and may be relevant to the possible development of antibiotic resistant infection in wounds and elsewhere.

(8) *Minor operation in a healthy patient.*

Here the anaesthetist need not be detained for long, but relative resistance to anaesthesia should be considered, and the premedication adjusted accordingly. Questioning on the lines outlined earlier in this paper should never be omitted, but if a satisfactory history is obtained, fitness for anaesthesia can usually be confirmed by a glance from the foot of the bed. Nevertheless, the basic examination as already described is nearly always desirable, partly to reassure the patient, partly for reasons of prestige and to prevent allegations of negligence, and also to check his story, and to ensure that no gross pathology is overlooked.

Out-patient anaesthesia. Anaesthesia must be brief and is virtually restricted to nitrous oxide and oxygen, although supplementation with a small amount of a volatile agent such as halothane need not unduly prolong the recovery time. If thiopentone is given, recovery may be prolonged, and the patient must never leave unescorted.

In a busy out-patient clinic, there is little time for comprehensive history taking and examinations. In spite of its apparently casual nature, the time-honoured custom of taking a rapid look at the patient as he walks in, and asking him one or two questions, is a remarkably effective method of assessment. Here too, it is assumed, that there is no certain method of forecasting the onset of coronary occlusion. Two distinct types of patient are, however, being sought, the unfit, and the very robust or resistant. Both are candidates for the predominant risk of nitrous oxide anaesthesia, prolonged and severe anoxia. The former, perhaps, because of an already impaired oxygen supply to an already damaged myocardium, the latter, because anaesthesia may not be attainable by nitrous oxide alone, without seriously restricting the oxygen intake as well.

The unfit are usually easily recognizable. Breathlessness, tachycardia, extreme pallor, cyanosis or uncontrollable coughing are signs which obviously arouse suspicion. Questioning to elicit exercise tolerance, the presence of chest pain or recent medical treatment will give further valuable information. The presence of congestive heart failure, gross cardiac or pulmonary disease can be confirmed on more thorough examination.

Since out-patients have usually arrived from work or home unaided, anyone whose condition arouses sufficient suspicion to warrant a full examination should be regarded as potentially unfit for nitrous oxide and oxygen anaesthesia, but in general it may be said that a patient who can walk up a flight of stairs without stopping and without distress, is fit for a short nitrous oxide and oxygen anaesthesia.

The resistant patient is encountered more frequently than the unfit out-patient. The very robust, the plethoric, the gross alcoholic and the highly nervous are easily recognized, but the very intelligent patient with a dynamic personality is often resistant to nitrous oxide and oxygen and his willingness to co-operate may easily lull suspicion. A history of previous out-patient anaesthetics should always be ascertained, and a story of struggling, absence of unconsciousness or of prolonged recovery are warnings that must not

be disregarded. Preparations to supplement the nitrous oxide should always be made for such patients so that adequate oxygenation can be ensured. The combination of a resistant personality with myocardial disease is particularly hazardous.

Potential obstruction of the upper airway should always be sought, and this is particularly important in the patient for nitrous oxide and oxygen alone. A short fat neck, trismus, oedema of the floor of the mouth or swellings of the neck usually presage a difficult anaesthetic, as do hoarseness or an old thyroidectomy scar (this should always be regarded with suspicion).

Most patients rejected for out-patient anaesthesia may usually be safely managed as in-patients, when premedication, pre-oxygenation and a smooth induction, perhaps with relaxant drugs, can easily be arranged. Whether the resistant patients are similarly treated will depend largely on the availability of recovery rooms, especially if thiopentone is used, and on the patient's domestic circumstances.

Children as out-patients are usually fit physically, and while the majority are amenable to sympathetic handling, a small minority, surprisingly difficult to forecast, resist all attempts to persuade them to inhale a gas or submit to an intravenous injection. The application of force in these circumstances can only be condemned, and although perhaps in a very few patients one may be tempted to "get it over quickly", it is usually preferable to postpone the attempt so that adequate premedication or basal narcosis may be given. It may even be desirable to admit the child to hospital, but this again will depend on domestic and other circumstances.

The risk of vomiting. As suggested previously, the usual rule of 4 hours pre-operative starvation may often be ignored for in-patients if they are anaesthetized and intubated with a cuffed tube in the head up position.

This technique, however, is usually less applicable to out-patients—unless adequate recovery facilities are available—so that stricter preparation is usually desirable, particularly for the nervous, the very obese or those with poor myocardial reserve who might not stand the anoxia associated with vomiting.

As there are situations, especially when a patient is in pain, where local analgesia is contraindicated and postponement is difficult or even undesirable on surgical grounds, the anaesthetist may sometimes decide that the risk of vomiting is acceptable. He must then be confident that should such an episode occur, he has adequate facilities to cope with it, and that the patient is able to stand an added anoxic strain.

CONCLUSION

This review of the factors to be considered by the anaesthetist is far from complete. No mention has been made, for example, of the special requirements of cardiac or neurological surgery. Little has been said about premedication or choice of anaesthesia, and only a few of the many possible intercurrent diseases have been mentioned.

The main points in routine assessment are however clear. In every case, the nature of the operation and the prevailing circumstances are of overriding importance. The anaesthetist must evaluate all the associated pathology and see that it is corrected as far as possible before operation. His decision is usually about the optimum time for operation rather than about its necessity. He must always look for potential difficulties with the upper airway and assess the likelihood of regurgitation or of vomiting.

He must pay special attention to respiratory disease and function when the operation is one which predisposes to chest complications, particularly if there is associated heart disease, but this is of much less importance before other operations. His most important duty at the pre-operative visit is to reassure the patient and calm his fears.

SUMMARY

The many factors to be considered at the pre-operative assessment are briefly reviewed.

A shortened form of clinical examination is described and the importance of taking a full history is stressed.

It is not possible to base an opinion on "fitness for anaesthesia" solely on the patient's clinical condition. Before any decision can be made, the latter must always be considered in relation to the nature and site of the proposed operation and to the prevailing circumstances.

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THE EVOLUTION OF PREMEDICATION

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THE history of sedation and the use of atropine in relation to anaesthesia has been recorded in detail by Duncum (1947). In outlining the history here, the changing use of drugs before anaesthesia to suit the anaesthetic, the currently fashionable anaesthetic techniques and the changing practice of anaesthetists, will be stressed.

The word premedication will be used throughout, though it was not employed in Great Britain until the late twenties before which "preliminary hyperdermic", "preliminary medication", or "pre-anaesthetic sedation" were used. The history falls naturally into two periods. The first lasted till 1910, and was marked by debate about the use of morphine and atropine, morphine and hyoscine, or atropine alone before chloroform. Thereafter morphine, atropine and hyoscine before ether became fashionable. This provided the basis for a real advance in anaesthesia, and formed the roots of modern premedication.

Until recent years premedication consisted of atropine or hyoscine, and morphine or Omnopon. The one inhibited salivation, the other sedated the patient. These are such obvious advantages that it is surprising to find that atropine was not used before ether to any extent prior to 1910. Indeed until then British anaesthetists were critical of the use of pre-anaesthetic morphine. The ascendancy of chloroform must be appreciated to understand this attitude to premedication, and it is against the background of this agent that opinions of British anaesthetists about the use of drugs before anaesthesia must be studied. Premedication before chloroform may be described under three headings—morphine or morphine and atropine before chloroform, atropine before chloroform (to prevent vagal inhibition of the heart) and morphine-scopolamine anaesthesia.

Morphine or morphine and atropine before chloroform.

This was seldom a premedication as we understand it for the comfort of the patient or the reduction of secretions was not being considered. Morphine was given in large dosages as part of the anaesthetic. The risk of chloroform appeared great but a morphine narcosis, supplemented by chloroform, would reduce the risk of chloroform by reducing the amount used. An early example of its use by Nussbaum (1864) is described as follows: " . . . He injected one grain of the acetate of morphia and the patient did not arouse, as is usually the case after chloroform inhalation, but remained in a most tranquil sleep during twelve hours. So sound was this sleep that even pricking with needles and knives and burning failed to arouse him."

At this time French physiologists, inspired by Claude Bernard (1869), were interested in anaesthetics and much about the early use of morphine and atropine with chloroform resulted from their work in animals. Claude Bernard, Professor of Physiology at the Sorbonne, practised premedication with morphine in his animal experiments. In 1869 he gave a series of lectures at the College de France on anaesthesia and on the use of morphine before chloroform. This led Labbé and Guyon (1872) of the Pitié Hospital to use morphine before chloroform in human surgery; in 1872 they described its use in four patients to the Académie des Sciences. In the same year Demarquay (1872) gave the Académie his experience of the method. He considered that it added to the dangers of anaesthesia and should be discontinued. Professor Dastre, another French physiologist, had assisted Claude Bernard in his anaesthetic researches. The fact that atropine prevented vagal slowing of the heart caused Dastre (1890) to use atropine with morphine in his animal experiments. To his dogs he gave atropine 1 mg (1/60 grain) and morphine 10 mg (1/6 grain) each per kg of body weight. This was

a morphine narcosis completed with a little chloroform. Dastre did not use atropine only to prevent cardiac arrest. He also recognized an antagonism between morphine and atropine, and that when the two drugs were given together the disadvantages of each were neutralized. The stimulating effect of atropine was controlled by morphine and the vomiting and respiratory depression caused by morphine were reduced by atropine. Forty years later, Dudley Buxton (1911b), championing the introduction of true premedication in Great Britain, employed the same arguments to justify his use of morphine, atropine and hyoscine. Dastre could not interest the Paris surgeons in his method. Eventually a Lyons surgeon, Aubert (1883), used it in human surgery in greatly reduced dosage. He gave atropine 0.5 mg (1/100 grain) and morphine 10 mg (1/6 grain) twenty to thirty minutes before chloroform and recorded its advantages as safety, rapid induction, quietness of the patient and reduced postoperative sickness. A contemporary of Dastre, interested in anaesthetics, was Dubois, Professor of Physiology in Lyons. Dubois (1894) did not favour morphine before chloroform, and (though recording that Aubert had obtained excellent results with Dastre's method) stated that recently it had been shown that deaths had occurred. The only impact of Aubert's work was in the practice of individuals. For example, Smith (1891), a London practitioner, recorded that since visiting the Continent and seeing Aubert's methods he had used morphine 10 mg (1/6 grains) and atropine 1 mg (1/60 grain) routinely. He then gave nitrous oxide followed by ether from a Clover's inhaler and stated: "Personally I am now in a great measure relieved from the painful anxiety attending the use of anaesthetics which an experience of twenty years had not diminished."

But surgeons and anaesthetists in Great Britain were generally critical of the use of morphine before anaesthesia and they are sometimes criticized for their reluctance to employ premedication. Sedation before chloroform could have reduced the risk of cardiac arrest and chloroform might have remained in use as an anaesthetic. In this criticism account is not taken of men like Hewitt and Dudley Buxton. If they were against the use of morphine before chloroform there was probably a good reason for their view. At present, when respiratory arrest is regularly produced during anaesthesia, it is not easy to realize the risk of giving respiratory depressant drugs before chloroform. In a Victorian theatre, respiratory arrest was almost as great a catastrophe as cardiac arrest is today. If the patient stopped breathing the surgeon started manual artificial respiration while the anaesthetist tried to maintain a clear airway. Any effective use of oxygen was unlikely. In such circumstances British anaesthetists were probably correct in thinking that morphine before chloroform was an added danger. Perhaps in one sphere only was it an accepted practice. Sir Victor Horsley (1886) advised its use before brain surgery. It reduced the amount of chloroform required in long operations and, what he regarded as more important, it markedly decreased bleeding. For a generation the weight of such an authority was reflected in the textbooks.

Atropine before chloroform to prevent vagal inhibition.

This probably represented the only routine use of true premedication in Britain during the chloroform era. Atropine was given before chloroform for forty to fifty years, probably quite uselessly; its value before ether was not generally realized.

Harley (1868), finding the pulse rate increased after atropine stated: "In all conditions in which there is a depression of the sympathetic nervous influence such as . . . failure of the heart's action from chloroform . . . the subcutaneous injection of atropia in doses of from 1/100 to 1/40 of a grain is the appropriate and most hopeful means of resuscitation."

When the pharmacology of atropine was better understood its use was advocated prophylactically. Atropine before chloroform involves us in the "chloroform problem". Why did healthy patients die during induction with chloroform? The cause was believed to be a reflex cardiac arrest. It was known that the vagus nerve slowed the heart and that atropine paralyzed the vagus. Anyone connecting these facts might suggest that atropine would prevent death under chloroform. The idea was put forward and practised by several who claimed it as their own. It was fostered by the observation that animals dying from cardiac

arrest under deep chloroform anaesthesia would not succumb if given a sufficient dose of atropine. One of the earliest recorded suggestions that atropine should be given in a routine fashion before chloroform was by Schafer (1880). ". . . It is well known that atropine paralyzes the cardiac inhibitory apparatus and since it is probable that death in these and similar cases results from a stimulation of this apparatus . . . there undoubtedly seems good reason for the employment of atropine. But, clearly it should be given immediately before administration of chloroform, *as a preventive.*"

From about 1870, Dastre (1890) gave atropine with morphine before chloroform in his experiments on dogs. He explained its use to prevent cardiac arrest: "Cutting the two vagus nerves would be the theoretical and brutal means of avoiding it. Such methods are inadmissible. There is, however, a practical and delicate method to arrive at the same result. It consists in the use of atropine. Atropine is equivalent to cutting the two vagus nerves."

As previously stated, Dastre gave atropine 1 mg (1/60 grain) per kg of body weight. The fact that so great a dose had to be given to annul vagal activity completely, prejudiced its use in human surgery and Dastre justified it only by combining it with morphine: "But atropine cannot be employed alone. It presents by itself certain dangers; it produces phenomena of excessive excitation . . . there exists against this drug a universal and irradicable prejudice."

Dubois (1894), who had criticized Aubert's employment of morphine before chloroform in human surgery, was equally critical of the use of atropine: " . . . It is only with great caution that it should be administered in clinical practice even associated with morphine."

Dudley Buxton (1909), with the same problem of dosage in mind, stated that the discomfort caused by the dose of atropine necessary to abrogate the vagal reflex rendered it almost unusable. Clinical doses were, however, given in man and by 1900 the use of atropine before chloroform was a common practice in Great Britain. But chloroform deaths continued and the value of atropine was questioned.

A milestone in the chloroform problem was the work of an Australian anaesthetist, Embley. He investigated cardiac arrest in dogs under chloroform in the physiological laboratory of Professor Martin of Melbourne University. In February 1902, Professor Martin addressed the Society of Anaesthetists in London concerning the results of Embley's investigation. Later, Embley (1902) published his work proving that the cause of death in morphinized dogs under chloroform was vagal inhibition of the heart. His findings were definite: "Section of the vagi or atropinization absolutely abolishes heart arrest under chloroform."

Embley gave no dosage of atropine and did not suggest its use prophylactically in clinical anaesthesia. A clinical dose of atropine would not be equivalent to vagal section. The less well informed, however, seized on Embley's researches as a justification for the use of atropine. A writer in the *British Medical Journal* (Editorial, 1902), reviewing Embley's work made it seem almost as his own suggestion. "The heroic remedy of cutting the vagi, though practicable in the dog, is naturally not to be recommended in man . . . atropine is a drug which quickly and temporarily paralyzes vagus endings in the heart and the subcutaneous injection of a small dose of atropine is clearly the correct treatment."

In 1901 the British Medical Association set up the Third Chloroform Committee. The report of 1911 accepted Embley's work as applicable to man and as proof that cardiac arrest under chloroform was due to vagal inhibition. Vagal inhibition, according to the report, occurred only when more than 2 per cent of chloroform was inhaled; no suggestion was made that atropine should be used prophylactically.

The next milestone in the chloroform problem was the work of Levy and Lewis appearing first in 1911 and culminating in the publication of *Chloroform Anaesthesia* by Levy in 1922. He held that cardiac arrest during induction with chloroform was ventricular fibrillation and criticized Embley's work on the ground that it was done on morphinized dogs. He wrote: "The liability of the heart to vagal inhibition was demonstrated in animals which had been very heavily dosed with morphia in addition to chloroform, 0.25 to 0.5 grammes of morphia, and the tendency of morphia to favour vagus effects is well known . . . and further, there can be no question at all, on the

evidence of Embley's own tracings, that the heart so inhibited is subject to recovery on the prompt application of restorative measures."

Embley (1915) did not accept Levy's work as being entirely applicable to man. His main criticism was that it was done on cats, which are peculiarly liable to ventricular fibrillation. He stated "There is no doubt as to the occurrence of ventricular fibrillation in cats. There is also no doubt about the absence or extreme rarity of its occurrence in dogs under chloroform."

Not only did Levy consider atropine useless, but he believed it increased the dangers of the induction period, although it could be of value where there was undue slowing of the heart during deep chloroform anaesthesia. He held that "There is little tendency to salivation under chloroform, so that the preliminary injection of atropine is unnecessary . . . the general use of atropine is in fact to be condemned for it is safer to retain the full action of the vagal inhibitory centre. Vagal tone diminishes the irritability of the ventricles; if vagal tone be abolished the ventricles are more ready to pass into an irregular tachycardia and subsequently to fibrillate."

The routine use of atropine before chloroform had for long been practised. As anaesthetists were discarding chloroform, atropine was suggested as a factor which had contributed to cardiac arrest! Although Levy's results closely resemble the observations on deaths in man during induction with chloroform, his conclusions were not universally accepted, and it was still held that death could occur from vagal inhibition of the heart.

Morphine and scopolamine before chloroform.

Morphine and scopolamine started off as a method of anaesthesia and not as a premedication. Its introduction is accredited to the German surgeon Schneiderlin. In 1900 he described the use of morphine and scopolamine in large dosage as an anaesthetic and in 1903 he published a second paper giving his experience of the method. He gave a test dose of up to 1 mg (1/60 grain) of scopolamine and 20 mg (1/3 grain) of morphine some days before operation and the necessary dose for surgery was gauged from this. It was given one and a half hours pre-operatively, and if anaesthesia was not complete, a further dose was given a quarter of an hour before operation. Schneiderlin (1903) described major surgery performed under morphine and scopolamine alone. Waters (1938) tried out the method and gave, over a period of two and a half hours before operation, 130 mg (2 grains) of morphine and 4 mg (1/15 grain) of scopolamine to obtain complete anaesthesia and relaxation. It is natural that at its inception the method was used with caution and that, with insufficient dosage, anaesthesia had to be completed with chloroform. Dirk (1904) stated that he purposely gave the drugs in reduced dosage, leaving the anaesthesia to be deepened with chloroform, and so something like premedication emerged. The use of morphine and scopolamine anaesthesia and morphine and scopolamine before chloroform achieved some popularity on the Continent and the literature on the subject became extensive. In this country, surgeons and anaesthetists had criticized the value of morphine before chloroform too long for the atmosphere to be favourable to the idea of the use of morphine and scopolamine before chloroform anaesthesia. During the first decade of the century individual anaesthetists used the drugs and wrote little. In 1908, at the annual meeting of the British Medical Association, the surgeon Macnaughton-Jones (1908) gave a paper on "scopolamine-morphine-chloroform anaesthesia" which he had used in twenty-eight patients. At the end of the talk his three anaesthetists, among whom was Dudley Buxton, gave a short account of their observations of the method as if to justify its use. Buxton stated that he was impressed by its value in nervous subjects, that the dangers were those familiar to surgeons who had employed morphine before chloroform, and that the utmost care was necessary to limit the amount of the chloroform used. Buxton was leaning towards premedication. In 1909, at the section of anaesthetics of the Royal Society of Medicine, he said: "The psychic element must not be forgotten; a terrified patient, a sleepless night. In such a patient I am convinced that the use of scopolamine and morphine before a general anaesthetic is valuable."

But he would not clash too vigorously with the accepted view. Sir Victor Horsley, in the same

discussion, said that few would agree to employ scopolamine and morphine generally, as a preliminary measure to the administration of a general anaesthetic. Buxton said he was at one with him as to the persistent dangers of morphine and explained he had in mind only its use under special circumstances. The anaesthetist was dependent on the surgeon, who employed him. The surgeon influenced the anaesthetic he used and the anaesthetist was at some pains to be in agreement.

The inception of true premedication.

The change from chloroform to ether preceded by morphine, atropine and hyoscine appears in the literature not as a transition but as sudden change. In 1910 chloroform seemed secure, ether was used by individuals and the use of premedication was criticized. By 1912 the era of chloroform was passing and all the talk was of the new advance in anaesthesia—atropine, morphine and hyoscine before ether.

The position in 1910 can be looked at briefly. The standard textbooks were those of Hewitt and Buxton, and a new edition of each appeared in 1907. Hewitt does not refer to scopolamine or morphine and scopolamine, though they had been in use since 1900. Buxton has a short paragraph on scopolamine. Both deal fully with morphine before anaesthesia, both are cautious, and Hewitt is definitely against its use. Neither suggest that atropine might be used before ether to inhibit secretions. In 1910 members of the Section of Anaesthetics of the Royal Society of Medicine discussed "The Choice of the Anaesthetic." There was critical comment by most speakers on the salivation and secretions caused by ether. Buxton (1910), dealing with anaesthesia in children, stated: "Some years ago I tested the effects of giving ether to young children and the main points which struck me were the liability to salivation and bronchorrhoea, which I regarded as not only distressing but injurious to the children." The use of atropine is not suggested; chloroform was advocated as the more appropriate anaesthetic: "So now my practice is to confine ether to brief operations even in middle life, and to follow it with chloroform."

The blindness of British anaesthetists to the value of atropine before ether, though they knew of its drying effect, was a result of the current method of administration—the closed inhaler. If atropine is to prevent secretions the induction had to be smooth. In 1910 the value of atropine before ether from closed inhalers was not apparent. The popular use of atropine to inhibit salivation, and of sedation to quieten the patient, were introduced at the same time, and to some extent the smooth anaesthetic from atropine before ether was attributed to the combined drugs. Later came the realization that the respiratory depression from sedation before open ether might make the attainment of relaxation more difficult. At the time, atropine, morphine, and hyoscine before open ether made it a practical substitute for chloroform. The secretions, cyanosis, and laboured breathing of the closed methods gave place to a smooth anaesthesia, comparable to chloroform, and, in average hands, it appeared to be safer. The practice was to some extent in advance of authority. Dudley Buxton dominated the scene, and until 1910 he was taken up with the work of the Third Chloroform Committee. His aim was to make chloroform safe, not to abandon it. Before 1910 he was critical of premedication. After 1910 he appears as the champion of its use and had much to do with the propagation of the new ideas. In March 1911, Buxton (1911a) gave a paper entitled "The use of Scopolamine, Morphine, Atropine and Similar Drugs before Inhalation Anaesthesia" to the Section of Anaesthetics of the Royal Society of Medicine.

In the discussion it is seen that each anaesthetist had only his own experience of premedication, and that there was no pooled knowledge. This meeting was like a release. A pent up interest in premedication was loosened and the tide had turned. Reports on the use of drugs before anaesthesia now began to appear frequently in the medical press. At first the emphasis was not on their use before ether particularly, but it quickly passed to the employment of morphine, atropine and hyoscine before open ether. Gilbert Brown (1911) in the *Lancet* wrote on "Three Hundred Cases of General Anaesthesia Combined with Narcotics". Felix Rood (1911) in the *British Medical Journal* described "Four Hundred Cases of General Anaesthesia Preceded by Scopolamine". In June 1911 the *Clinical Journal* in-

included a lecture by Buxton (1911b) on "Methods of Combining the Use of Alkaloidal Substances and General Anaesthesia". This was a full review of the subject, historical and clinical. Buxton advocated a mixture of morphine, atropine and hyoscine. He said that, given together, morphine and hyoscine produced a sedation achieved only by a dangerous dose of morphine. Atropine counteracted the respiratory depression of morphine, reinforced the feebler drying effect of hyoscine and lessened the inhibitory action of the vagus. Another drug which became fashionable in premedication at this time was Omnopon. It had been marketed on the Continent in 1909 under the name Pantopon and its introduction could not have been better timed. In the first article on its use in Great Britain, Leipoldt (1911) described it as the ideal pre-anaesthetic narcotic.

The use of premedication in the years before the 1914–18 war caused the same interest as the use of curare in our time. Speaking at the Section of Anaesthetics of the Royal Society of Medicine, Rood (1912) said that the progress of general anaesthesia had recently been marked by two considerable advances. The first was that the inhalation of 2 per cent chloroform was practically free from danger and: "The second great step in the development of anaesthesia was the discovery that certain narcotics and atropine could be combined with open ether to produce an anaesthesia which had all the advantages of chloroform and none of its dangers and gave, all things considered, results decidedly better than those of chloroform."

In 1912, Buxton was President of the Section of Anaesthetics of the British Medical Association at its annual meeting in Liverpool. The section discussed "The Use of Alkaloidal Bodies Prior to Inhalation Anaesthesia" (*Brit. med. J.*, **2**, 620). In 1913 the 17th International Congress of Medicine was held in London. With Buxton as President, members of the Section of Anaesthetics discussed "Alkaloidal Bodies alone and in Conjunction with General Anaesthesia" (*Lancet*, **2**, 556). At the same meeting Teter of Cleveland talked of the practice of using nitrous oxide and oxygen in major surgery. The first of his principles was the use of pre-anaesthetic medication.

In 1913 the section of Anaesthetics of the Royal Society of Medicine held a second discussion on premedication. Compared with the 1911 discussion it is evident that pre-operative drugs were now in regular use. McCardie (1913) of Birmingham expressed views which later became accepted practice. He gave morphine and hyoscine to all except the very old and the very young to whom he gave atropine alone. He said that morphine and hyoscine checked secretions sufficiently without the use of atropine.

The wave of enthusiasm lasted for a further year or two. In 1915 members of the Section of Anaesthetics of the Royal Society of Medicine held a third discussion on the use of pre-operative drugs. It was opened by Blomfield (1915), and Hewitt, Boyle, Barton, Mennel and Shipway took part. The use of ether had become general and all were unanimous as to the value of premedication. The safety and peculiar value of atropine alone before ether was recognized. In distinction to morphine and hyoscine, for which individual treatment was advocated, it was suggested that atropine could be used in a routine manner. Hyoscine came in for criticism. With premedication followed by open ether, surgeons were encountering a rigidity they were not accustomed to under chloroform. Hyoscine was blamed and Mennel remarked that his surgeons were wont to call a rigid abdomen a "scopolamine belly". In the same discussion Boyle told of his use of preliminary drugs at a base hospital: " . . . before this injection was given, the men, although fairly quiet at the start of the induction, almost invariably struggled and fought. But now all this was changed. They came up in a drowsy condition and were anaesthetized quietly and quickly."

Falling in with the fashion of the time, the work of Crile (1913) was a stimulus to the use of premedication. Writing in the *Lancet* on anoci-association he advocated morphine and hyoscine before operation for the prevention of shock. Meanwhile, in the United States, where surgeons had not become accustomed to the relaxation provided by chloroform, anaesthesia was developing in other ways. The very light open ether used by the Mayo brothers at Rochester was described by Berry (1912): "No preliminary drug was used except in stomach cases in which 1/6 grain of

morphine is usually given. . . . The patient is fairly firmly strapped on the table. In abdominal cases the anaesthesia was kept light throughout. At the first incision there was usually some slight movement and sometimes inarticulate sounds on the part of the patient. As a rule the surgeon took no notice. While the stomach itself was being operated on the anaesthesia was usually stopped altogether and the patient often came round enough to speak."

She commented on the excellent results. Her opinion was confirmed that light anaesthesia was safe anaesthesia and rendered the patient less liable to shock, but she said that the depth of anaesthesia frequently considered sufficient at the Mayo clinic, would not satisfy a good many surgeons over here. In Chicago she saw morphine and scopolamine anaesthesia employed for major surgery: "The anaesthetist sat by and gave a little chloroform when required. The patient appeared quite conscious most of the time, but it was stated there never is any recollection of pain during operation."

The Section of Anaesthetics of the Royal Society of Medicine had discussed premedication in 1911, 1913, and 1915. The furore then died down and it was 1928 before "Preliminary Medication in Anaesthesia" was again considered.

With the exception of the standard works by Hewitt and Buxton the new ideas were slow in appearing in the textbooks. A fourth edition of the small textbook *Anaesthetics* by Blomfield (1917) illustrates the quality of anaesthesia available when ether was used from a closed inhaler without premedication: "Secretion of mucus and saliva takes place fairly freely during the early stages of ether taking, and to some extent throughout. A noisy gurgling sound with respiration may be caused by mucus in the upper air passages and trachea."

Dealing with open ether in children he wrote: "The ether excites a good deal of secretion which must be wiped away from time to time. In spite of the gurgling sound of the respiration no bronchitis follows short administrations. For long operations upon children, 1/200 grain of atropine may be given with advantage."

Hesitancy to include the new ideas arose from a doubt as to whether or not premedication was a passing fashion. In the earlier discussions it had been pointed out that this was no new thing; it had been practised long ago in France, had fallen out of favour, until it returned with the introduction of hyoscine. Interest at that time might soon have subsided. Marshall (1917), dealing with anaesthetics in a casualty clearing station, commented on the danger of excessive dosage with morphine in severe limb injuries: "It is my experience that a badly injured patient has a poor chance of rallying if he has received more than a quarter of a grain of morphine before operation."

Marshall gave no atropine to patients with abdominal wounds as he had been unable to discover any advantages from it.

Hyoscine too was still criticized but for an additional reason. It had acquired a reputation for unreliability. Buxton (1911b) had mentioned this: "It has been pointed out by a large number of observers that the actions of scopolamine and hyoscine appear to differ, and that a sample of scopolamine or hyoscine may differ from another sample according to the source from which it is prepared."

This reputation of hyoscine, which persists to some extent to the present day, had its roots in the infrequent revision of the British Pharmacopoeia. Hyoscine was not included in the 1898 edition and anaesthetists first used it as a non-official drug of varying composition. The next revision in 1914 included hyoscine, but as it was not realized that the sedative effect was achieved by laevo-hyoscine, no polarization test was included. Meanwhile the German Pharmacopoeia had described hyoscine as laevo-hyoscine and called it scopolamine, which was recognized as a dependable product and used by anaesthetists in place of the official drug. The British Pharmacopoeia of 1932 described hyoscine as laevo-hyoscine, but surgeons, physicians and anaesthetists alike continued to prescribe the the "continental" scopolamine.

By 1920, though the word premedication was not yet in use, and sedation before anaesthesia was by no means completely accepted, atropine before ether was an established practice. Machines were appearing to replace open methods and the old authorities were passing. Hewitt had

died in 1916 but his textbook, revised by Robinson reached its fifth edition in 1922, and dealt fully with the use of pre-operative drugs. Dudley Buxton had retired in 1919 and the sixth and final edition of his textbook was published in 1920. Here too pre-anaesthetic medication was fully described and its routine use advocated. Ether had replaced chloroform as the anaesthetic of choice and the value of atropine in promoting its use was pointed out. "Ether is the best and safest anaesthetic for general purposes alike for adults and children. . . . Whenever ether is to be given a preliminary injection of atropine should be given, provided no contra-indication is present. When this plan is adopted most of the objections formerly advanced against the use of ether disappear."

In Great Britain premedication had evolved as a technique of anaesthesia, a means of giving a smooth ether anaesthetic and a way of escape from chloroform. From now on other factors such as the comfort of the patient, the reduction of metabolism and the prevention of shock and post-operative vomiting were to have more effect on its continuing evolution.

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CORRESPONDENCE

HALOTHANE AND CARDIAC ARREST

Sir,—The paper by Dr. V. Torry Baxter (*Brit. J. Anaesth.*, **32**, 4, 1960) describing a cardiac arrest following administration of a high concentration of halothane vapour raises a number of interesting points apart from its principal contribution, which draws attention to a mechanical failure of the Fluotec vaporizer.

Firstly, there is the question of undue reliance upon instruments rather than sound clinical assessment of the patient. Long before well calibrated instruments were available we were taught that irrespective of the settings of a flowmeter or the predetermined amounts of drugs to be injected, the only sure guarantee of safe anaesthesia was the reaction of the patient to the drugs being administered. Consequently flow rates etc. were adjusted to the patient and not the patient to the flowmeters. I have been disturbed that more and more this very sound principle is being ignored and it is assumed that all is well because a vaporizer has been set to a certain concentration, or that because a ventilator has been set at certain pressures, rates and rhythm the patient of necessity must be well ventilated. Nothing is further from the truth. Had Dr. Baxter followed the patient's blood pressure instead of merely recording the temporal pulse he would presumably have noted at 0922 hrs. that there was marked hypotension and he would then have reduced the halothane concentration or completely shut off the vaporizer. Experiments carried out in our laboratories and published prove beyond a shadow of a doubt that monitoring of the pulse rate alone is quite inadequate in halothane anaesthesia.

The second point which gives food for thought is equally basic. Here was a patient who had generalized diminution of chest movement on respiration. This is an important observation, but is it sufficient reason to modify the anaesthetic management for a short procedure, such as a herniorrhaphy, in such a way as to make a relatively simple, straightforward anaesthetic a major and complicated procedure, requiring the administration of a multitude of drugs, viz. thiopentone, suxamethonium, topical lignocaine, nitrous oxide, pethidine and halothane? Obviously intubation and intermittent positive pressure change the entire management of the anaesthetic. I venture to say that a good old-fashioned anaesthetic with nitrous oxide, oxygen and ether, following thiopentone induction, would have been a good deal safer even in the presence of diminution of chest movement, having regard to the stimulating effect of ether upon respiration. If the surgeon could not be prevailed upon to dispense with the cautery (and why should he not have made concessions in the interest of the patient's well-being?), a low spinal or epidural block would have been in order. Alternatively a good local anaesthetic would have been highly satisfactory for an inguinal hernia repair. I do not wish in any way to discount the importance of the diminution of chest movement in this patient, but in all matters medical one risk must be weighed against the other. It is not permissible, by adjusting to one circumstance, to introduce a number of other factors which in their aggregate are likely to be much more hazardous than was the original condition which the change of technique was designed to circumvent.

I believe, Sir, that these are considerations of the greatest importance and I would make a plea for return to simplicity in those instances where simplicity is possible.

GORDON M. WYANT,
University of Saskatchewan, Canada

Sir,—Professor Wyant makes two reasoned criticisms of the clinical technique described in my paper (*Brit. J. Anaesth.*, **32**, 171) and like myself, subscribes to the views put forward by Nosworthy (1953a, b).

It is not easy to avoid being wise after the event. I am in complete agreement with the principle that the whole process of anaesthesia must be tailor-

made to the individual patient: there is, in fact, no such procedure as a "routine anaesthetic", and recognition of this raises us above the level of being mere handmaidens of the surgeons. Had I known of the vaporizer's bizarre performance on two previous occasions, I would not have used the apparatus: had I not been specifically asked to allow the use of diathermy, I would (probably) have used nitrous oxide, oxygen and ether—even though the operation was to be performed, under instruction, by a junior surgeon whose time for the herniorrhaphy was not likely to be less than 1¼ hours.

Given the requirements of diathermy and long anaesthesia for the particular patient, and not being granted second sight, I do not consider that the original intention was any worse a choice than some form of regional anaesthesia. The intention had been simply (I use the word deliberately), to produce unconsciousness, analgesia, and relaxation in the presence of adequate respiratory ventilation. I did not expect to have to give more pethidine (after the 20 mg i.v. injection at 0915), nor did I anticipate being compelled (by the attempted spontaneous respiration at about 0917) temporarily to introduce halothane. In condensing the clinical record, blood pressure readings were not included in the paper: at 0900, 0910, 0915 and 0920 the systolic readings were respectively 135, 125, 130 and 125. Working single-handed, I accepted the visible evidence of absence of active bleeding from the wound and the surgeon's statement (both at 0924) that femoral arterial pulsation was not palpable, and got on with my part in the resuscitation manoeuvres instead of taking a blood pressure reading.

Professor Wyant rightly observes that the considerations which he puts forward are of the greatest importance. In teaching, I also constantly emphasize the fact that, unlike patients, apparatus and drugs are standardized but flexible in our hands.

V. Torry Baxter
Bristol

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